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with the kind regards
of the Author

THE
PHYSICAL DIAGNOSIS
OF
THE LUNGS AND HEART.

LECTURES

ON THE

PHYSICAL DIAGNOSIS

OF THE

DISEASES OF THE

LUNGS AND HEART.

BY

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TO

FREDERICK COBB, ESQ., M.D.,

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS;
SENIOR PHYSICIAN TO THE LONDON HOSPITAL;
ETC., ETC.

These Lectures are Inscribed,

BY

HIS SINCERE AND ATTACHED FRIEND,

THE AUTHOR.

PREFACE.

In publishing the following Lectures, which were originally delivered at the Royal Infirmary for the Diseases of the Chest, the Author has had a two-fold object in view:—first, to collect and arrange, as concisely as possible, for the use of the student, the facts which relate to the Theory and Practice of Auscultation and Percussion; and secondly, to direct the attention of his medical brethren to the labours of the distinguished school of Vienna, of which Professor Skoda is the acknowledged head and founder.

Although considerable attention has been devoted in these Lectures to an examination of the principles advocated by this original observer, the special aim of the Author has been to treat the subject in a manner essentially practical, and to introduce the most important investigations which have been recently made in this interesting branch of Physical Diagnosis. For this purpose, the observations of the most eminent British and Foreign authorities have been consulted, and compared with the results of the Author's own experience in his public and private practice.

The author may be allowed to state, that the portion of this course, which was published in the 'Lancet' of 1850, has been translated into the German language by Dr. Albers, Professor of Medicine at Bonn, to serve as the groundwork of a text-book for the students of that University.

In conclusion, the Author takes this opportunity of thanking Dr. Andrew Smith, Director-General of the Army Medical Department; Mr. Henry, Principal Medical Officer at Chatham; his colleague, Dr. Little; Mr. Hovell, of Clapton; and Mr. Hacon, of Hackney; who kindly afforded him every facility in the prosecution of his inquiries—especially in those investigations which relate to the nature and causes of the Venous Murmur.

A few diagrams have been introduced, for the drawings of which the author is indebted to Mr. Brushfield, of the London Hospital.

23, *Finsbury Square;*
Oct. 1851.

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DISEASES

OF

THE LUNGS AND HEART.

LECTURE I.

STATISTICS—PHYSIOLOGY.

The Official Report of the Registrar-General, which returns the number of Deaths annually occurring in England and Wales, and the causes to which the mortality is ascribed, presents abundant evidence of the importance and absolute necessity of the study of the Diseases of the Lungs and Heart.

Thus, if we take into consideration the mortality of the Metropolis, we find that the number of deaths from all causes amounted, in 1845, to 48,475; in 1846, to 49,277; and, in 1847, to 60,442; of which those ascribed to the Diseases of the Chest amounted, in 1845, to 16,356; in 1846, to 16,151; and in 1847, to 20,583; and those to Pulmonary Consumption, in 1845, to 6731; in 1846, to 6890; and in 1847, to 7010.

Hence, the ratios of the number of deaths resulting from diseases of the chest and pulmonary consumption to the entire mortality of the metropolis respectively were, in 1845, $\frac{1}{2\cdot9}$ and $\frac{1}{7\cdot2}$; in 1846, $\frac{1}{3\cdot0}$ and $\frac{1}{7\cdot14}$; and in 1847, $\frac{1}{2\cdot9}$ and $\frac{1}{8\cdot6}$.

With reference to the year 1847, it must be borne in mind, that the deaths from bronchitis, pneumonia, and pleurisy, very far exceeded in number those of many preceding years, for, on examining the details of the Registrar's Report, we find that the excess of mortality in 1847 over 1846, due to those diseases, amounted respectively to 1912, 1740, and 99, while the excess from phthisis pulmonalis, for the same period, rose only to 120. This will account for the smaller ratio which the number of deaths from consumption bears to the entire mortality for that year.

Again, if we take into consideration the entire population of England and Wales, we find, that in the year 1841, when the Census Commissioners returned the population at 15,912,773, the entire mortality amounted to 343,847, of which the deaths ascribed to diseases of the chest amounted to 96,729, and those to pulmonary consumption to 59,592.

Hence we may safely conclude, that the number of fatal cases due to the affections of the respiratory and circulating organs amounts almost to one third of the entire mortality of the country, and that nearly one sixth of the deaths occurring in any year are referable to pulmonary consumption. In addition to the information respecting the relative frequency of thoracic disease, I am anxious to draw your attention to the periods of life at which these affections are found to be most destructive. For this purpose, I must again draw upon that mine of information, the Report of the Registrar-General, a document which, from the mass of data it presents, is worthy of the especial attention of every one interested in the important subject of medical statistics. The following table exhibits the relative frequency in the metropolis of the most important diseases of the lungs, arranged quinquennially, and according to the data supplied for the year 1846. To make the comparison complete, I have included hooping-cough and croup, diseases classed by the registrar with those of the zymotic order.

(1846.)

	Hooping-cough.	Croup.	Laryngitis.	Bronchitis.	Pleurisy.	Pneumonia.	Asthma.	Phthisis.	Total.
At the age of 5 years and under	2025	275	83	888	19	2480	7	683	6460
,, ,, 10 ,, and above 5	4	...	...	5	3	26	...	165	203
,, ,, 15 ,, ,, 10	2	...	...	11	3	31	3	447	497
,, ,, 20 ,, ,, 15	...	...	1	21	8	46	4	749	829
,, ,, 25 ,, ,, 20	...	...	2	29	8	36	4	884	963
,, ,, 30 ,, ,, 25	1	...	2	45	6	47	9	839	949
,, ,, 35 ,, ,, 30	...	...	4	51	14	47	21	855	992
,, ,, 40 ,, ,, 35	...	1	6	67	17	59	33	702	885
,, ,, 45 ,, ,, 40	1	...	3	98	15	60	45	557	779
,, ,, 50 ,, ,, 45	...	...	6	123	13	42	82	369	635
,, ,, 55 ,, ,, 50	...	...	2	135	8	59	99	278	581
,, ,, 60 ,, ,, 55	...	...	4	196	10	57	131	164	562
,, ,, 65 ,, ,, 60	...	...	3	229	5	64	127	114	542
,, ,, 70 ,, ,, 65	...	...	2	223	10	39	123	51	448
,, ,, 75 ,, ,, 70	...	...	2	152	5	28	75	20	282
,, ,, 80 ,, ,, 75	...	...	...	104	2	18	30	3	157
,, ,, 85 ,, ,, 80	...	...	1	35	...	9	8	1	54
,, ,, 90 ,, ,, 85	...	...	...	15	...	1	...	...	16
,, ,, 95 ,, ,, 90	...	...	...	3	...	...	...	...	3
Ages uncertain	2	1	...	1	...	2	1	9	16
Total	2035	277	121	2431	146	3151	802	6890	15853

Although, for the sake of compression, I have omitted a few diseases, the number of deaths from which are too small to affect the general reasoning, and have also purposely excluded a class vaguely returned as "diseases of the lungs," we have sufficient evidence presented by the table to show:—

1. That at least two fifths of the deaths from diseases of the lungs occur at and under the age of five years.
2. That croup and hooping-cough are peculiarly fatal to infancy.
3. That pneumonia is also extensively fatal in early life, nearly 80 per cent. of the deaths from that disease occurring at and under the age of five years.
4. That the deaths from bronchitis are most frequent at and under the age of five years, being nearly in the proportion of 33 per cent., and that above that age and under twenty-five, the mortality is at a minimum, increasing from that period upwards.
5. That 10 per cent. of the deaths from consumption occur at and under the age of five years; that the maximum of mortality is between fifteen and forty years of age, amounting to nearly 60 per cent. of the entire number of fatal cases from this disease—a statement which closely accords with the experience of the Medical officers of the Hospital at Brompton, as given in their valuable Report for 1849.

The facts which thus prove the dangerous character of the diseases of the chest, exhibit but in part the absolute frequency of these affections; for while, from the nature of the Report, no mention is made of the non-fatal cases of catarrh, influenza, and other pulmonary disorders, which at some seasons of the year appear to sweep over the length and breadth of the land, it excludes from the list of deaths ascribed to diseases of the chest many cases of apoplexy, dropsy, rheumatism, and other diseases which have their origin in, or at least owe their fatality to, some morbid condition of the lungs or heart.

With such statements as these, I believe that it would be occupying time to little purpose to dwell upon the importance of the subject of these Lectures. I shall content myself with simply remarking that no one at the present day can engage in the practice of his profession, with safety to his patient, or credit to himself, without having previously become intimately acquainted with the means of detecting the numerous forms of disease to which the organs in the chest are liable. Let me also impress upon you the fact, that no species of intuition, no preconceived notions, can afford any idea of the physical signs derived from auscultation and percussion. However versed you may be in the morbid anatomy of the thoracic viscera, and however thoroughly you may comprehend the theory of the normal and abnormal sounds audible in the chest, your knowledge will prove to be utterly valueless unless combined with persevering clinical experience. Examine case after case as patiently and as accurately as possible; trace out the changes which the sounds undergo from day to day, and observe how disease gradually passes away, and gives place to the natural healthy condition of the organ. Or, should the case terminate unfavorably, be careful to examine the *post-mortem* appearances, and thus familiarise yourselves with the physical causes which led to the production of the abnormal sounds heard during your investigation of the patient. A few months thus employed will go far to make you experienced auscultators, and afford the means of detecting a class of diseases which, until the time of Laennec's great discovery, was involved in absolute gloom and uncertainty. With these preliminary remarks, I shall at once proceed to the proper object of the course, and occupy the remainder of this Lecture with a brief sketch of the anatomy and physiology of the contents of the thoracic cavity.

The chest is a large conoid cavity, the parietes of which are composed of bone, cartilage, ligament, and muscle, so admirably adjusted to each other, that strength and lightness of structure, wonderful facility of motion, and security to the important organs contained in it, result from the union of these parts. Its boundaries are the sternum and costal cartilages in front, the seven true and five false ribs on each side, and the dorsal vertebræ behind. Its floor, formed by the irregularly convex surface of the diaphragm, presents three important openings,—one between the crura of the lesser diaphragm, for the passage of the aorta, thoracic duct, and vena azygos; a second formed by the decussation of the crura for the œsophagus and par vagum; and the third in the tendinous centre of the muscle, for the transmission of the inferior vena cava.

The upper orifice of the cavity is a plane, which represents the section of an obliquely truncated cone; so that, in consequence of the position of the clavicles, the apparent differs very considerably from the real form of the upper outlet of the thorax. The diameters are,—the perpendicular, the antero-posterior, and the transverse. The first, by reason of the obliquity of the upper and lower orifices of the chest, has its maximum length along the vertebral column, and its minimum represented by a line drawn from the centre of the upper orifice to the middle of the arch of the diaphragm. The antero-posterior has its greatest length between the xiphoid cartilage and the opposite dorsal vertebra, while the lateral or transverse diameter is longest at the level of the ninth or tenth ribs, and gradually diminishes towards the apex of the chest. I pass hurriedly over the ordinary anatomy, as I suppose you are already familiar with its details. The muscles by which this framework of bone and cartilage is put in motion, and rendered subservient to the process of respiration, are divided into those of inspiration and expiration, each of which may be ordinary, forced, or violent. The ordinary respiratory movement in men is for the most part performed by the action of the diaphragm lengthening and shortening the perpendicular diameter, the intercostal muscles taking but a small share in the ordinary expansion of the chest. The respiration of females is, however, more costal and less diaphragmatic than that of men; the number of their inspirations may be easily counted by watching the movements of the bosom, whilst the same fact is best obtained in men by placing our hand upon the abdomen, and observing the rise and fall of its walls. Physiologists have been struck with this remarkable difference between the male and female chest, and have assigned various reasons for its existence. Some have ascribed it to the peculiarity of female dress, supposing that compression of the waist impedes the expansion of the lower part of the chest, restrains the action of the diaphragm, and consequently throws a greater amount of work upon the ribs. This view, although, to a certain extent, undoubtedly true, as I shall have occasion to show, when speaking of Dr. Sibson's spirometer, cannot be entirely adopted, for the same peculiarity of respiration is found in females of all countries, and is observed also in young girls whose forms are free and not yet victimised to fashion. Other writers have explained the fact by supposing that Nature, anticipating the growth of the gravid uterus, and its probable effect upon the downward movement of the diaphragm, has guarded against any evil consequences which might result from this condition, by endowing the female with a greater comparative power over her intercostal muscles. I believe that this view is not very improbable. Whatever be the true explanation, it cannot be denied that the framework of the female chest is more delicately constructed than that of the male, and that she consequently possesses a greater elasticity and mobility than the male.

The muscles concerned in forced and violent respiration may be exemplified by the description of a case of excessive dyspnœa. The head and neck are thrown back and fixed by the aid of the muscles inserted into the occipital region; the scalenus anticus and medius muscles, arising from the transverse processes of the cervical vertebræ, and inserted into the first rib, co-operate to fix that bone; the second rib, being also secured by the contraction of the scalenus anticus, forms a *point d'appui* for the action of the intercostal muscles, which elevate and turn the bodies of the ribs outwards, and in this way enlarge the horizontal and antero-posterior diameters. The scapula, being fixed by the trapezii, rhomboidei major and minor, and levatores anguli scapulæ muscles, become fixed points for the lesser pectorals, subclavius, and serratus magnus muscles; and when the dyspnœa becomes excessive, the arms are thrown upwards and fixed, by the patient firmly grasping some support near him; and thus the pectoralis major and latissimus dorsi are enabled to lend their powerful assistance to the general expansion of the thoracic cavity.

Ordinary expiration is the natural return of the chest due to the weight and elasticity of its walls; the diaphragm becomes relaxed, and ascends into the chest; the abdominal muscles, which had been protruded by the descent of the diaphragm, return to their former position; the costal cartilages, which had been rendered tense by the inspiratory effort, now bring their elastic power into play, and aided by the resiliency of the lung, combine to produce a general diminution of the thoracic cavity. The forces engaged in forced expiration I shall merely enumerate, being the intercostales, abdominales, triangulares sterni, serrati postici inferiores, longissimi dorsi, sacro-lumbales, and quadrati lumborum, aided, as in ordinary expiration, by the natural elasticity of the ribs and cartilages, and the resiliency of the tissue of the lung.

From this sketch it will be seen that the inspiratory is entirely muscular, while the expiratory is partly muscular and partly elastic power. This naturally leads to a comparative estimate of the forces respectively exerted in the acts of inspiration and expiration,—a subject of considerable importance, as certain theories, in explanation of some morbid structural changes in the lung, have been founded upon a relation supposed to exist between them. By an instrument constructed by Dr. Hutchinson, ('Medico-Chirurgical Transactions,' vol. xxix,) the powers of inspiration and of expiration were measured, and the conclusions obtained found to be at total variance with the views previously entertained upon the subject.

"The resistance afforded to measure the true respiratory power was a column of mercury," and "the communication was made by a peculiar adaptation to the nostril, that the true respiratory muscles only may be tested; for were it by the mouth, the muscular act of smoking or suction by the tongue would interfere, and render the observation useless."

The results of at least 1500 experiments showed that while the power of the inspiratory muscles could raise a column of mercury two inches and a half, the force of the act of expiration was equivalent to a column of three to three inches and a half in height; and that in all cases the expiratory was at least one third greater than the inspiratory power. Let us observe how this balance in favour of the act of expiration can be explained.

By an instrument similar to a force-pump, but provided with a tube in such a way that the air driven into any vessel might return and react upon a column of mercury, Dr. Hutchinson was enabled to force a quantity of air into the chest of an individual (of course dead), and to measure the resistance which the elasticity of the lungs and ribs offered to its entrance. The subject operated on was a man,—height, five feet eight inches; weight, ten stone ten pounds; absolute capacity of his chest, 245 cubic inches; internal superficies of his chest, 250 square inches; area of diaphragm, fifty square inches; temperature of the body, 97° Fahr. This man had been tested during life by the spirometer (an instrument hereafter to be described), and had been found capable of inspiring 200 cubic inches of air. With these data, the problem to be solved was to determine the amount of the resistance offered to the entrance of 200 cubic inches into the lungs, a force evidently involuntary, and simply due to the opposing elastic power of the lungs, ribs, and cartilages.

The results were as follow :—

	Cubic Inches.			Inch of Mercury.
When	70	were forced in,	the resistance	= 1·00
„	+ 20	„	„	= 1·50
„	+ 90	„	„	= 3·25
„	+ 20	„	„	= 4·50

Whence we conclude, that the opposing elastic power of one square inch of the walls of the chest at the end of a full inspiration is equal to the weight of a column of mercury, whose base is one square inch, and height four inches and a half. And as the superficies of the chest (excluding the diaphragm) was 200 square inches, we find by the law of the pressure of fluids, that the whole elastic power of the lungs, ribs, and cartilages, at the end of a full inspiration = weight of a column of mercury, whose base is the superficies of the chest, and height = four inches and a half = 200 × 4½ cubic inches of mercury = 900 cubic inches of mercury = 450 pounds, nearly; from which we infer, that the inspiratory act of this man when alive, and at the end of an inspiration of 200 cubic inches of air, was opposed by a force of 450 pounds in the shape of elastic resistance, and that the expiratory muscular force was aided by the whole amount of this power. This excess of the expiratory over the inspiratory force will at once explain the fact of the normal expiratory occupying a much smaller period of time than the inspiratory murmur. Before concluding this part of the subject, I would draw attention to the fact, that the figures given in the above experiment very aptly account for the quick and frequent respirations which are made by an individual whose general muscular system is in a weakened condition. It will be seen that the elastic resistance is small in amount at the commencement of an inspiration, but increases in a very rapid ratio with the additional quantity of air inspired. In the case before us, the resistance for the first seventy cubic inches was represented by a column of mercury of one inch in height; but with an increase of 130 cubic inches of inspired air, the resistance rose to four inches and a half of mercury. Hence, it is evident that the muscular system of the thorax would attempt in a debilitated condition to work under the smallest pressure possible, and effect, by several small inspirations, what, under other circumstances, would have been accomplished in a single effort. The respiration of the young infant exhibits in a remarkable degree the effect of the resisting elasticity of the chest. "The breathing is quick and short, then after a few seconds there succeeds a pause, and then the hurried respiratory movements begin again, while the slightest disturbance on the most trivial excitement will at any time raise the frequency of the inspirations by ten or twelve in the minute. This respiration, too, is almost entirely abdominal; the chest moves but little, and its walls are but little expanded; and the ear detects little or nothing of that clear sound which is characteristic of a subsequent period of childhood, and with which you are all familiar by the name of puerile respiration. This peculiarity of the breathing in early infancy, to which M. Trousseau was, I believe, the first to call attention, is another token of the feebleness of the inspiratory power. As the child grows older, and its strength increases, and its muscular system becomes more developed, the chest expands with each inspiration, and the faint respiratory murmur is succeeded by the loud puerile breathing which is heard as the air enters into the small air-cells." (Dr. West, 'Lectures on Diseases of Children.') The nerves which are principally concerned in the respiratory movements are the par vagum and sympathetic, as incident or excitor, and the phrenic, intercostal, spinal accessory, long thoracic or external respiratory of Bell, and those branches of the spinal nerves supplying the abdominal muscles, as motor. It would be out of place to track the course of the several nerves above mentioned. I would only remark that the par vagum, by its superior and inferior branches, is a regulator of the glottis, the janua vitæ, and that its ultimate branches being distributed over the stomach, point to the connection existing between dyspnœa, dyspepsia, and functional derangement of the heart. Lastly, the origins of the phrenic nerves must be borne in mind in reference to spinal injuries. Thus, as the nerve arises from the third, fourth, and fifth cervical nerves, any severe injury above the third will produce instant death by suddenly paralysing the diaphragm and intercostal muscles; while if it occurs below the sixth vertebræ, the patient may live for some hours, if not days, although the action of the greater number of the intercostal muscles must be wholly or partially arrested. Those who are students of the London Hospital will remember the three cases of fractured spine which had been lately received into that establishment; each of the sufferers complained of a feeling of intolerable pressure when breathing (compared by one to half a ton weight)—a sensation evidently resulting from the partially paralysed intercostal muscles being incapable of fully opposing the elastic power of the lungs and ribs, the resistance of which we have calculated to amount, at the end of a full inspiration, to two pounds and a quarter upon every square inch, or to nearly 450 pounds upon the whole superficies of the chest.

The lungs are irregular conoid bodies, the bases of which rest upon the diaphragm, while their apices fill the upper part of the chest, and project somewhat above the clavicles. Their form, varying with their degree of inflation, is generally convex on their outer and lateral surfaces, and concave on the inner surface, and their bases are more or less plano-concave, according to the degree to which the diaphragm is contracted. Between the fourth and fifth ribs, and near the left edge of the sternum, a lozenge-shaped space is left between the two lungs, where a portion of the heart remains uncovered, the remainder of that organ being received into a depression in the inner surface of the left lung. Each lung is divided into two large lobes by a fissure passing from the back part of that organ downwards, and forwards to the anterior angle of its base. The right is further divided by another septum, which traverses the lung from the middle of the former fissure to the anterior edge of that organ. The right lung is shorter than the left, in consequence of the position of the liver, but its breadth is somewhat greater, so that (with some exceptions) the right side measures half an inch more than the left. The two lungs are connected by the root, which consists of the primary bronchi, the pulmonary and bronchial arteries, the pulmonary plexus of nerves, which sink into the substance of the organs, while the pulmonary veins pass out to pour their contents into the left auricle. Each lung is covered by the pleura, a fine serous membrane, which, being reflected from the pulmonary surface over the internal parietes of the chest, forms a shut sac, whose sides, in consequence of the presence of a thin exhalation, glide smoothly, and without noise, over each other at every movement of respiration. Between the two sacs so formed is a space arbitrarily divided into the anterior, middle, and posterior mediastinum, for the contents of which I must refer to the ordinary anatomical works. This sketch of the contents of the thoracic cavity would be evidently imperfect without a description of the minute anatomy of the pulmonary organs—a knowledge of which is absolutely essential for the due comprehension of the nature of the morbid changes to which the structure of the lung is liable, and of the rationale of the physical signs resulting. The subject is one of peculiar interest, and has of late years occupied the attention of the best microscopic observers. Among these Messrs. Rainey and Quekett stand most prominent, and to the latter gentleman I am indebted for the drawings and beautiful specimens illustrative of this portion of our subject. The paper to which I refer, and upon which I have drawn largely, is one by Mr. Rainey, published in the 'Medico-Chirurgical Transactions' for 1845, and which clearly and concisely describes the minute structure of the lungs.

According to this writer, the lung-parenchyma is composed of—

1. *Bronchial tubes.*
2. *Bronchial intercellular passages.*
3. *Air-cells.*
4. *Plexuses of vessels and nerves.*

The whole invested by the thin serous pleural membrane.

1. The *bronchial tubes* which divide and subdivide in the substance of the lung, become gradually diminished in caliber, and terminate rather abruptly within one eighth of an inch of the pleural surface in the bronchial intercellular passages, their diameter at this point varying from one fiftieth to one thirtieth of an inch. Their constituent parts are cartilaginous rings, or portions of rings, and mucous membrane; the former gradually disappearing with the progressive ramifications of the bronchi, till the smallest tubes are reduced to the condition of simple membrane. The mucous lining is firmer and thicker in the upper part of the respiratory tract, presenting circular (organic muscular fibres, according to Kirkes and Paget,) and longitudinal fibres from the primary bronchial division downwards to the point where the intercellular passages

commence. Its entire surface is covered with vibrating ciliated epithelium. The bronchial arteries are the nutrient vessels of the mucous membrane and general parenchyma of the lung. Springing from the aorta, they distribute branches in their course to the pericardium, bronchial glands, and œsophagus; and reaching the air-tubes, follow the subdivisions of these channels until they ultimately anastomose with the vessels of the lung cells: the fact that the terminal branches of the bronchial arteries do not enter the true vesicular texture of the lung, is one of considerable importance, and bears upon the differential pathology of bronchitis and pneumonia. The ultimate distribution of these vessels, and the mode in which their blood is returned to the heart, has been at all times involved in some obscurity. Ruysch and Cowper believed that the terminal branches of the bronchial arteries united directly with the capillaries of the pulmonary artery; while other observers, like Reisseisen, maintained that the blood of these vessels passed at once into the pulmonary veins, without being exposed to any further action of the atmospheric air. Harrison informs us, that in a few instances the injection passed from the bronchial into the pulmonary artery, but that in no case was he able to fill the bronchial from the pulmonary artery. Dr. Graves confirms this view to some extent; and I think we may conclude that the two systems of vessels are almost entirely distinct in distribution, termination, and function; and that while the bronchial arteries discharge almost the whole of their blood into the bronchial veins, into the vena azygos, the entire contents of the pulmonary artery pass on to the left heart through the four pulmonary veins. These considerations, although apparently barren of interest, are really of considerable importance, as they elucidate the pathology of the whole subject of hæmoptysis.

2. The *bronchial intercellular passages* are the natural continuations of the air tubes, and differ from the latter in being destitute of mucous membrane and epithelium; they are, in fact, simply passages around which the air-cells cluster. At their commencement, these channels are cylindrical in form; but in their progress towards the pleural surface they assume an irregular shape, running between the vesicles, and communicating with them in all directions, and ultimately terminating in a single air-cell, the form of which in no way differs from the rest.

3. *Air-cells* or *lung-vesicles* are irregularly-shaped cavities, which vary in size in different parts of the lungs, being smaller but more vascular in the central than in the peripheral portions of those organs. Being situated close upon the bronchial tubes, or intercellular passages, they open into them by large circular apertures, the borders of which are sharp and distinct in outline. Where no intercellular passages exist, the cells are observed to open into each other—a sacculated arrangement, easily seen by altering the distance of the object glass of the microscope from the object, and bringing different planes into view. The pulmonary membrane forming these quadrangular-shaped cells is excessively thin and transparent, and is composed of fibres which, according to Mr. Rainey, have no resemblance whatever to either kind of muscular fibre. It is unprovided with epithelium, and is totally

distinct from the membrane which lines the bronchial tubes. It has diseases peculiar to itself, and inflammation of the cells formed by it runs a different course to inflammation of the bronchial mucous membrane, a result evidently depending upon the different natures of the two structures.

4. The *plexuses of vessels* are situated immediately beneath the pulmonary membrane, in such a way that the blood is merely separated from the atmospheric air contained in these cells by the interposition of this delicate structure. And this exposure is so fully ensured, that the capillaries between the cells are aërated on both sides, being enclosed in the fold of membrane which forms the sides of contiguous cells. We can form some idea of the wonderful mode in which these capillaries are packed together, when we find that, according to Kirkes and Paget, the interspaces or meshes are narrower than the vessels themselves, the diameters of which are, on an average, not more than $\frac{1}{3000}$th of an inch.

From this description of the minute anatomy of the lungs, we conclude that these organs are nothing more than the vast expansion of a fine, delicate membrane, upon the opposite sides of which blood and atmosphere are situated; and that the entire mechanical arrangements we have followed out are simply intended to ensure those agents being brought together in such a manner, that by their mutual chemical reaction the black blood of the pulmonary artery may be converted into the bright-red blood of the pulmonary veins. This is the sum and substance of the process of respiration;

and whenever any impediment exists which more or less excludes the air from the lung-cells, or any cause is in operation to produce an inordinate accumulation of blood in the capillary plexuses of the pulmonary artery, Nature attempts, by increased inspiratory efforts, to balance the disproportion between the air and the blood. Should the attempt be attended with some difficulty, a peculiarly distressing sensation results (dyspnœa); or if the balance can be in no way restored, death from suffocation is the consequence (apnœa).

This is a very brief summary of the doctrine of Dyspnœa; but I hope again to revert to it.

LECTURE II.

SPIROMETRY.

In my last Lecture I sketched out so much of the Anatomy and Physiology of the Chest as seemed sufficient to make my future remarks intelligible. I drew attention to the framework of the thorax, the mechanism by which it is put into motion, and the ordinary and minute anatomy of the lungs, and concluded by touching cursorily upon the nature of dyspnœa.

I must now proceed to the special object of this course,—the means by which thoracic diseases are to be detected,—which are:

1. Spirometry.
2. Inspection.
3. Mensuration.
4. Palpation.
5. Succussion.
6. Percussion.
7. Auscultation.

1. Spirometry.—This method of determining the permeability of the lungs to air is by no means of recent date. It was employed many years ago, under the designation of pulmometry; but in consequence of a total disregard to the height, weight, and age of the individuals examined,—circumstances which essentially modify the capacity for inspiration,—results were obtained of so contradictory a character, that the method was soon abandoned, as leading to no conclusions upon

which any reliance could be placed. A valuable paper, however, has been communicated by Dr. Hutchinson to the Medical and Chirurgical Society, which promises to place the subject on a proper footing with respect to diagnosis, and to lead the way to a more correct application to spirometry. The views contained in this communication ('Medico-Chirurgical Transactions,' vol. xxix) are of such interest and importance, and derived from so extended a series of observations, that I consider myself compelled to lay before you a short analysis of the paper.

Almost every writer on the physiology of respiration has attempted to determine three circumstances:—

1. The quantity of air capable of being contained in the chest.

2. The quantity of air employed in ordinary respiration.

3. The quantity of air which can be inspired by the strongest voluntary effort.

But the statements published have been so discordant that little value can be attached to the results obtained by former observers. In proof of this assertion, I need only refer to the conclusions obtained by Abernethy and Reid, who respectively calculated the number of cubic inches of air requisite for ordinary respiration to be 12 and 100. The discrepances found in the results arrived at by former experimenters, in making spirometrical observations, may be accounted for by the fact, that no regard was paid to Height, Weight, or Age; for we shall find hereafter that, if these circumstances be taken into consideration, certain rules may be deduced, by which we are enabled, *à priori*, to determine the number of cubic inches of air a healthy individual should inspire, and, conversely, the number of cubic inches expired being given, the height may be determined approximatively, provided the individual be in a sound condition at the time of his examination by the instrument. This statement may appear strange, but it is undoubtedly correct, being the result of at least 3000 experiments upon individuals in all classes of society.

As the chest is continually changing in capacity, its movements may be divided into those of—

1. Extreme expansion.
2. Extreme contraction.
3. A state intermediate to the two.

And as the quantity of air contained in the chest is always changing, it may be arranged under the four following heads :

(1.) The residual air; or the quantity remaining after extreme expiration, and which no voluntary power can expel from the lungs.

(2.) The reserve air; or the quantity of air left after an ordinary expiration, *minus* the reserve air.

(3.) The breathing air; or the quantity between ordinary inspiration and ordinary expiration.

(4.) The complemental air; or the quantity taken into the chest, after an ordinary inspiration, by a forced effort.

(2), (3), and (4), are termed by Dr. Hutchinson "tenants at will," being liable to be turned out at a moment's notice. The quantity represented by (2), (3), and (4), collectively, is termed the "vital capacity," and is evidently the whole amount of air taken into the chest by the fullest inspiration possible.

The instrument by which the vital capacity is determined is termed the "Spirometer." Dr. Hutchinson has given, in his paper, a plate of the instrument he is in the habit of employing; but I prefer a modification introduced by Dr. Pereira, which is, in my opinion, more simple, and less liable to error. The instrument is, in fact, nothing but a small model of a gasometer. It consists of a large glass cylinder, suspended, by means of a cord, in a reservoir of water,—the cord passing over a pulley, and having a weight attached, so that, by proper adjustment, the cylinder may balance in any position. A pipe, forming the continuation of the tube through which the patient has to breathe, rises in the bell-glass above the level of the water; and by forcing the air through this tube, the vessel will ascend, and indicate, by a graduated scale affixed, the quantity of air passed into it. The principle of the machine is very simple, and its use easily acquired. It will be found, upon experiment, that the number of cubic inches capable of being expelled from the lungs, after a full inspiration, by an individual in good health, is liable to such little variation, that the vital capacity may be considered as a constant quantity. I have tried my own respiratory powers very many times, and find that my vital capacity in no degree differs from its amount three years ago.

I shall not occupy your time with all the tables and elaborate calculations made by Dr. Hutchinson, but proceed at once to state, that the vital capacity depends upon four circumstances:—

1. *Height of the individual.*
2. *Age.*
3. *Weight.*
4. *Disease.*

1. *Height.*—Strange as it may appear, the height of an individual has been incontestably proved to be the chief condition which regulates his vital capacity, so that, as a general rule (subject to a few corrections), we may determine the height of a healthy individual from a knowledge of the number of cubic inches of air which he is capable of expiring. It might be supposed that this result depends upon the length of the body, but the most casual observation will assure us of the fact, that differences of height depend principally upon the length of the legs, and not of the body. Dr. Hutchinson at first imagined that the absolute capacity of the chest altogether decided the amount of the vital capacity, under the supposition that tall men had proportionally larger chests than short men. On carefully examining a great variety of chests, and observing their circumference, breadth, and depth, and even measuring the absolute capacity of several individuals after death, by taking casts of their thoracic cavities, he found that the size of the chest bore no necessary relation to the height of the individual, and that while many tall men had very small chests, and many short men very large chests, their vital capacities always remained proportionate to their heights. It was from

observing this discrepancy that he was led to take height into consideration. With respect to the reason of this relation, our author says,—"I confess myself as much at a loss to explain it as I was the first day I commenced the research. I believe the vital capacity is mathematically commensurate with the mobility or thoracic movement; but why the mobility increases in arithmetical progression with height, which appears chiefly dependent on the length of the limbs, and not on the length of the trunk of the body, I am incapable of explaining. So completely is mobility, and consequently the vital capacity, affected by stature, that a man will breathe in different positions different quantities of air: thus, standing, I blow 260 cubic inches; sitting, 255; and when recumbent, (supine) 230, (prone) 220;—position making a difference of forty cubic inches." The latter statements, I believe, are easily explained, if we bear in mind the weight and elasticity of the chest walls, and the unfavorable condition which the recumbent position offers for the play of the respiratory muscles. The relation of the height to the vital capacity is, however, to me equally as mysterious as to Dr. Hutchinson. The following table, deduced from the observation of a vast number of healthy individuals, exhibits the vital capacity for all heights between five and six feet; and it will be seen, as a rough rule, that for every additional inch in height, there is a corresponding increase of eight cubic inches in the vital capacity:—

Height.	From Observation.	Regular Progression.
Ft. In. Ft. In.	Cubic Inches.	Cubic Inches.
5 0 to 5 1	174	174
5 1 „ 5 2	177	182
5 2 „ 5 3	189	190
5 3 „ 5 4	193	198
5 4 „ 5 5	201	206
5 5 „ 5 6	214	214
5 6 „ 5 7	229	222
5 7 „ 5 8	228	230
5 8 „ 5 9	237	238
5 9 „ 5 10	246	246
5 10 „ 5 11	247	254
5 11 „ 6 0	259	262

2. *Weight* forms an important element in the calculation, although its effect has not yet been fully determined, from the circumstance that the standard weight of an individual of a given height and age remains undecided. To elucidate this point, Dr. Hutchinson, with an undaunted perseverance, obtained the weights of 2648 males at the middle period of life, and calculated the mean weight for each height. The following was the result:—

Height. Ft. In.	Mean Weight. lbs.
5 1	119·9
5 2	126·1
5 3	132·9
5 4	138·6
5 5	142·1
5 6	144·6
5 7	148·4
5 8	155·2
5 9	162·1
5 10	168·6
5 11	174·2

The comparison of the results given by the spirometer, with the weights of individuals, proved, that for the height of five feet six inches a man may exceed his standard weight (as shown in the last table), without suffering any diminution in the amount of his vital capacity, but that with every increase beyond that point, the quantity of air inspired was diminished, in the ratio of one cubic inch, nearly, for every additional pound in weight. Thus, if we suppose a man to weigh 10 stone, or 140 pounds (his height being five feet six inches), he may increase by 7 per cent., or by 10 pounds, nearly, without any loss in his vital capacity, while any addition to 150 pounds will be marked by a diminution in the quantity of air which his chest is capable of inspiring.

3. *Age.*—We should naturally expect the inspiratory power to share, with the other functions of the body, in the vigour which characterises the adult period of life. Experiment confirms this view. The vital capacity is found, *cæteris paribus,* to be at a maximum between the ages of thirty and thirty-five, and to fall gradually after the latter period, at the rate of nearly one and a half cubic inches for every additional year.

4. *Disease.*—We have now arrived at the most important element of variation, for the detection of which the spirometer is, of course, intended. It is quite evident, that if it is possible to determine, *à priori,* the standard vital capacity for all heights, weights, and ages, that the instrument puts us in the possession of a means of deciding upon the normal or abnormal

condition of the respiratory organs of any person subjected to an examination by it. This is the basis of all our views respecting the spirometer; and the 3000 experiments made by Dr. Hutchinson provide the standard of health in question. It is true that in many cases the instrument is inapplicable, and could only be employed at the risk of injuring the patient. Bronchitis, pneumonia, and many of the pulmonary affections, present such distinct and significant signs of their existence, by auscultation and percussion, as render the employment of spirometry entirely useless; but there is a disease, insidious in its approach and fatal in its consequences, in which the powers of the instrument become eminently serviceable. Every medical practitioner of any experience, in the observation of phthisis pulmonalis, must be fully aware of the difficulty frequently found in many cases, of determining with precision upon the existence of the early stage of this disease. At this period the local signs are often too few, or too obscure, to enable us to decide, with certainty, upon the real nature of the complaint, however warranted we may be in suspecting the existence of pulmonary consumption, from the observation of the general and functional signs. It is at this point that the spirometer comes in to our assistance, and I must admit that I have frequently found considerable value in the indications which it exhibits. My own experience readily brings to my recollection numerous instances where the ordinary means of investigation have failed to satisfy my mind, and where the difficulty has been greatly removed by an examination, by the spirometer, of the respiratory powers of the patient.

From the observation of many hundred cases of suspected tubercular disease of the lungs, I have learned that a marked diminution of the vital capacity is an important indication of the disease in question, and that this condition of the inspiratory power is always antecedent to such a manifestation of the local signs, as would be sufficient to warrant us in giving a decided opinion upon the existence of the affection. The spirometer is simply an auxiliary—it does not and cannot supersede the usual modes of examination, but is valuable at a time when these are insufficient. Its utility is therefore somewhat limited in range: but the particular disease in which it is more especially serviceable is, unfortunately, so common, and the necessity of recognising the early stages of the affection so important, in a therapeutical point of view, that the profession cannot but feel grateful to Dr. Hutchinson for the manner in which he has brought the subject of spirometry before their notice. Independently of its value in assuring us of the existence of the early stages of pulmonary disease, the negative results (if I may so call them) of the instrument are equally of importance. I have examined many individuals whose apprehensions respecting their thoracic organs have been completely dissipated by finding that their vital capacity was equal to the normal—the simple knowledge of the fact being sufficient in itself to remove their hypochondriacal condition and to effect an almost instantaneous cure. I am aware that several objections have been raised to this mode of investigation, and that many members of the profession believe the true vital capacity cannot be obtained by reason of fear, nervousness, and other causes supposed to impede the free action of the respiratory muscles. A little practice with the Spirometer will, however, convince that these objections are ill founded; some degree of care and attention being alone requisite to prove the correctness of the statements which have been advanced respecting the utility of the instrument. As an interesting example of the subject, I quote the case of Freeman, the well-known American giant, who was at one time an out-patient of the London Hospital. Previous to this period he had been examined by Dr. Hutchinson, who found the following to be his measurements:—

Height, seven feet; weight, nineteen stone five pounds; circumference of chest, forty-seven inches; vital capacity, 434 cubic inches. Two years subsequent to this period, he was taken ill, and, although complaining of some dyspnœa, no trace could be found at the time of the deposit of tubercular matter in his lungs. "An examination by the spirometer, however, showed that his vital capacity had sunk to 344 cubic inches. In a year from that time he had fallen a victim to consumption." This case fully indicates the value of the Spirometer; and by the experience derived from an extended series of observations made with it, I have come to the conclusion that a very small amount of tubercular deposit is sufficient to produce a great diminution in the natural elasticity of the lung, and a corresponding reduction in the vital capacity.

The following are a few cases taken from my note-book, showing the alterations in the amount of air respired in certain diseased conditions of the pulmonary organs:—

	Age.	Height.	Vital Capacity.	Normal Vit. Cap.	
		Ft. In.		Cub. In.	
A . . .	24	5 9	190	238	Phthisis, 1st stage.
B . . .	35	5 8	104	230	Phthisis, 2d ,,
C . . .	21	5 8	150	230	Phthisis, 2d ,,
D . . .	19	5 10	230	246	General debility.
E . . .	38	5 3	110	190	Pneumonia.

These examples will show the mode in which the examination is to be made. A, B, C, were undoubted cases of consumption; D presented the general and some of the functional signs of that disease, but ultimately recovered his strength and health; E was an instance of chronic consolidation of the lower half of the right lung, which had continued during a period of at least three years.

Spirometry, by Sibson's Chest Measurer.—I have again to draw attention to the 'Medico-Chirurgical Transactions,' for a paper by Dr. Sibson, on a new mode of determining the mobility of the chest. The instrument, termed the chest measurer, and invented by the author of this valuable communication, is a species of spring, which, when applied to the parietes of the chest, measures the movements of its diameter, and "indicates, by the motion of the index hand on a dial, any movement of respiration to the hundredth of an inch." It is, in reality, a pocket spirometer, and enables us, with great facility, to measure the space through which the chest walls move during ordinary or forced respiration.

Male Adult.—The mobility of the sternum and six upper ribs, ranges in ordinary respiration from ·02 to ·06 inches; and in extraordinary, from ·5 to 2 inches.

The mobility of the eighth, ninth, and tenth, (diaphragmatic ribs,) ranges, in ordinary respiration, from ·05 to 1 inch, while in forced respiration it is less than that of the upper ribs.

The advance of the abdomen, in ordinary respiration, at a point midway between the ensiform cartilage and the umbilicus, is from ·25 to ·35 inches; and, at the sides, from ·08 to 1·2 inches.

Hence it is evident, from these figures, that the costal movements are very slight in ordinary respiration, being less than one sixth of the abdominal, and that the lower ribs take a larger share than the upper in common tranquil breathing.

The greater elasticity of the frame in youth renders the costal respiration more marked at that period of life than subsequently; while in old age, when each cartilage becomes more or less ossified and rigid, the respiration is carried on to a greater proportional extent by the diaphragm.

Female.—Compression of the waist by stays exaggerates the movements of the thoracic, and restrains those of the diaphragmatic ribs.

When the stays are on, the motion at the second ribs ranges from ·06 to ·2 inches; the motion of the diaphragmatic ribs, from ·06 to ·11 inches: while, when the stays are off, the motion at the second ribs ranges from ·03 to ·1 inch; the motion of the diaphragmatic ribs from ·08 to ·2 inches; and the deeper the inspiration the greater is the disproportion between the range of movements with and without stays. I have previously alluded to the difference in the respiratory movements of the male and female chests, and I find Dr. Sibson states his belief, that even if females wore no stays, "the thoracic respiration would be greater, and the diaphragmatic less, than in men."

The respiration in children is more costal and less diaphragmatic than that of adults, in consequence of the greater proportionate size of their abdominal organs, and the greater softness and elasticity of their ribs and cartilages. The abdominal movement ranges from ·06 to ·15 inches; the thoracic expansion, from ·02 to ·12 or ·15 inches.

Having obtained the standard measurements of the respiratory movements in males, females, and children, we are now in a position to detect the differences which result from any causes tending to impede the costal or diaphragmatic movements. These causes may be general or partial, may affect one side entirely, or only a portion of it, and in some instances their effect may even produce, during the act of inspiration, a depression instead of an elevation of a part of the chest wall. Thus, when the diaphragm acts suddenly and forcibly, and the quantity of air inspired is disproportionate to the space left by the movement of the muscle, the lungs partially collapse at the lower part of the chest, and the parietes corresponding become partially flattened by the external atmospheric pressure. This result is found to occur in the crowing inspiration of children, pertussis, croup, hysteria, and, to a certain extent, in bronchitis and emphysema.

The chest-measurer, while it indicates, like the spirometer, the existence of some cause which impedes the free movement of the thorax, affords no direct evidence of the exact nature of the affection. Thus, on referring to the "causes which may restrain the costal and diaphragmatic respirations of one side, those of the opposite side being exaggerated," we find that the chest-measurer may indicate any of the following affections :—

An obstruction in a large bronchus.
Emphysema and bronchitis of the whole of one lung.
Pleuritic effusion and pneumothorax of one side.
Condensation of the whole of one lung from effusion.
Consolidation from phthisis, combined with cavities and tendinous adhesions of the whole of one lung.
Pneumonia.
Extensive external injury, (fractured ribs.)
Pleurodynia.
Lateral curvature of the spine.
Hemiplegia (?).

Whence we must conclude, that the value of the instrument, as a means of diagnosis, is limited.

Dr. Sibson, at the conclusion of his paper, states that "the knowledge furnished by the deranged movements and rhythm of respiration defines the seat of the disease, but not its nature: to ascertain this, the other aids to diagnosis must be employed. This knowledge is the first step to the inquiry, which it does not prolong, but, on the contrary, shortens, as it directs the attention to the affected part." My own experience with the instrument has, however, led me to the conclusion, that the chest-measurer has been of greater utility in determining the rationale of certain physiological and pathological points, respecting the thorax and its contents, than in assisting us in the diagnosis of the diseases to which the lungs and heart are liable.*

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* Since the delivery of the above remarks, Dr. Richard Quain has invented an ingenious instrument called a Stethometer, which enables us to measure, with great facility and accuracy, the amount of expansion capable of being exhibited by any portion or the whole of the chest. "It consists of a case like a watch-case,—on its upper surface is a dial, and also an index. This case contains a simple movement by means of which the index can be acted on. A silk cord which acts on the index passes through the aperture in one side of the case.......It is evident, that if the instrument be so placed that extension be made on the cord, the amount of the extension will be shown by the movement of the index on the dial. For example, if the instrument be laid flat on the spine, and held in its place by the first and second fingers of the left hand, whilst the cord is carried round the chest, and pressed on one of the ribs on the sternum by the fingers of the right hand, then when the individual under examination expands the chest during inspiration, the amount of expansion will be communicated to the cord and thus indicated on the dial. The cord may then be directed around the opposite side of the chest, and thus, will be at once seen any difference which exists in the relative mobility of the two at the point under examination." A full account of the instrument is given by Dr. Quain in the 'London Journal of Medicine' for October, 1850.

LECTURE III.

PERCUSSION.

An accurate acquaintance with the relative positions of the organs contained in the cavities of the thorax and abdomen is absolutely essential as a preliminary to the study of Percussion. By careful anatomical research, the student should render himself intimately acquainted with the boundary lines of the several viscera; he should be able to recognise the prominences and depressions upon the surface of the body which correspond to certain parts of the subjacent organs, and which, serving as guides to their position, enable him, at a glance, to map out the limits of the several contents of the chest and abdomen. Having mastered the geography of the interior, he should familiarise his senses of hearing and touch, by the frequent percussion of the body, with the sound and sensation of resistance presented by the various regions subject to examination, and thus prepare himself to detect those alterations of position and density, which individually, or in combination, accompany the diseases of the thoracic and abdominal organs.

The act of percussion is, in itself, a simple operation, and, perhaps, so simple, as not to have engaged your attention; it will not, therefore, be out of place to bring before your consideration, in a concise manner, the circumstances and conditions under which sound is elicited by the percussion of bodies. You are aware that the sensation of sound is produced by a body whose molecular particles are in a state of vibration, and a medium by which these vibrations may be communicated to the delicate apparatus of the ear, and so made to impress the auditory nerve. Without vibrations we have no sound; but it does not follow conversely, that where vibrations exist, sound must inevitably be produced. In addition to the vibrating body we must have a medium also capable of being set in vibration—thus, if a bell be struck, the vibrations of its metallic particles set the surrounding particles of the air into vibration, and the air becomes the medium of communication between the bell and the ear. Now, if the bell be placed under the receiver of an air-pump, and the air exhausted, though the bell be struck, and vibrations produced, no sound will be heard, in consequence of the absence of a conducting medium; but in proportion as the air is readmitted into the receiver will the sound caused by the bell become more audible. Again, although a body may be in a state of active vibration, and the vibrations be communicated to a medium capable of transmitting them to the ear, the sensation of sound will not be produced, if the vibrations are less than 32, or if they exceed 8200, in a second of time. This power of molecular oscillation possessed by different bodies, depends upon the elasticities of those bodies, different degrees of elasticity producing different capabilities of originating and conducting sound.

"But an important condition in the constitution of bodies is homogeneity of structure; and in a substance perfectly homogeneous, we may add, too, uniformity of structure. The effect of want of homogeneity in a structure, on its power of propagating sound, is precisely analogous to that of the same cause in obstructing the free passage of light, and for the very same reason. The sonorous pulses, in their passage through it, are, at every instant, changing their medium. Now, at every change of medium, two things happen:—first, a portion of the wave is reflected, and the intensity of the transmitted part is therefore diminished; secondly, the direction of propagation of the transmitted part is changed, and the sonorous rays, like those of light, are turned aside from their direct course. Thus, the general wave is broken up into a multitude of non-coincident waves, emanating from different origins, and crossing and interfering with each other in all directions. Now whenever this takes place, a mutual destruction of the waves, to a greater or less extent, arises, and the sound is stifled and obstructed. Further yet, as the parts of a non-homogeneous medium differ in elasticity, the velocities with which they are traversed by the sonorous pulses also differ; and thus, among the waves which do arrive ultimately at the same destination, in the same direction, some will arrive sooner, some later. These, by the law of interference, tend mutually to destroy or neutralise each other." (Sir J. Herschel, article "Sound," 'Encycl. Metrop.')

These principles will be sufficient to explain the nature of the doctrine of Percussion.

In accounting for the various modifications of sound, we have two conditions to bear in mind:—

1. The character of the vibrations dependent upon—

   (*a*) The violence of the force or impulse by which the molecules of the vibrating body are set in motion, upon which depends the *loudness* and *intensity* of the sound.
   (*b*) The number of equal vibrations in a given time, from which results the pitch or elevation of the tone.
   (*c*) The number of times these vibrations are repeated, by which the *duration* of the sound is regulated.
   (*d*) The nature of the vibrating body, upon which depends the quality or timbre of the tone.

2. The medium in which the vibrating body is placed.

Leaving these considerations for a time, our first inquiry is to determine the source of the sound obtained by the percussion of the chest.

The substances upon which the force of percussion is exerted when the chest is subjected to this mode of examination, are:—

1. The skin and muscle.
2. The bone and cartilage.
3. The parenchyma of the lung.
4. The air contained in the cells and bronchial passages.

That the sound is not due to the vibration of skin and muscle is evident from the dull, fleshy tone obtained from the percussion of the thickest portion of the thigh; nor does it result from skin, muscle, and bone, as will be observed by striking the palm of the hand, or parts covering the ilium. The parenchyma of the lung is, in itself, a very weak vibrating body, as is proved by the *post-mortem* percussion of compressed lung, whence we conclude that the principal source of the sound elicited by the percussion of the healthy chest, is the air contained in the cells and bronchial passages; and we infer that the object of our percussion is, to set this air into vibration, and to deduce its quantity and position from the character of the tone produced.

The sound will be found to depend upon three principal circumstances:—

1. The force of the stroke.
2. The quantity of the air below the part percuted.
3. The nature of the media upon which the percussion is effected.

1. The impulse by which the particles of air are made to vibrate, and the consequent loudness of the sound produced, will depend upon the force of the stroke, while the fulness and duration of the sound will be dependent upon the quantity or volume of air thrown into a state of vibration, and the total number of these vibrations.

2. The larger the quantity of air thrown into vibration, the fuller will be the character of the resulting tone.

3. The greater the elasticity, and the more homogeneous and uniform in structure the media, by means of which the effects of the stroke are conveyed from the centre of percussion to the volume of air contained in the cells and bronchial passages, the more easily will the volume of air be set in vibration, and the more readily will the resulting sound be propagated in return through the same media to the ear.

We can now understand how any cause which diminishes the quantity of air in the chest, or removes the lungs from the parietes, will tend to weaken or alter in tone and quality the sound resulting from percussion. There has been at all times considerable difficulty in assigning terms to sensations, and most of all to sounds. I shall adopt the divisions laid down by Professor Skoda, of Vienna, who arranges them into—

1. A full tone, and its opposite, an empty tone.
2. A clear tone, and its opposite, a dull tone.

By fulness is meant a considerable volume of sound: thus, a large cathedral bell even when muffled and vibrating weakly, conveys the idea of a large mass in motion, while a small hand-bell, however distinct its tinkling tone may be, cannot be mistaken for one of a large size; our ideas of size, as derived from sound, depend upon the degree of the fulness of tone.

By clearness is meant a sound which results from strong, well-marked, and non-interfering vibrations.

Empty and dull sounds I need not stop to define, as they are necessarily the respective antipodes to the above.

We may have combinations of these conditions.

1. A full and clear tone.

As in a large mass, vibrating strongly, and surrounded by a good conducting medium. This sound is obtained from well-developed chests, whose walls are elastic and but slightly covered with fat or loose fleshy substance. A drum gives the sound in perfection, and we find it well-marked in pneumo-thorax, where air has found its way into the cavity of the pleura.

2. A full and dull tone.

As in a large mass, vibrating, but having its vibrations damped. This is the tone of the muffled drum, of a fat and flabby chest. A small quantity of fluid in the cavity of the pleura will not prevent the vibration of a large quantity of air, but will materially weaken and muffle the sound produced by the most forcible percussion.

3. An empty and clear tone.

A sound which results from the strong vibrations of a small quantity of air. It may be easily obtained by inflating the cheeks, and practising percussion upon them when so distended. We have an excellent example of the tone in small superficial cavities, which are not only free from fluid, but bounded by firm walls, capable of reflecting the vibrations of the contained air.

4. An empty and dull tone.

This is the flesh sound, the Schenkelschall, or thigh-tone of Skoda, as the best example is afforded by the percussion of that portion of the body. It is met with in cases of consolidation, or compression of the lung.* "The quantity of fluid required to produce a thigh-tone, at any part of the thoracic or abdominal walls, depends upon the elasticity of the parietes under the spot percuted, and the nature of the space behind the fluid. If the spot is elastic, there is no air present within six inches of the surface. We can convince ourselves of this in the following manner:—Dip a portion of lung, or intestine, which contains air, under water, and percute upon the surface of that fluid by means of a plessimeter; the sound of the air in the lung or intestine will not be perceived when they are dipped more than six inches below the surface; but the nearer they are brought to the surface of the water the fuller will be the sound produced." The dullest sound obtained from the percussion of the chest arises from the presence of a large quantity of fluid in the pleural cavity.

Tympanitic Tone.—This sound, as its name indicates, resembles the tone obtained from a drum. It is never met with in the healthy chest, and is not always present when its chief condition, the existence of a large quantity of air in the pleural cavity, is present. Experiment would seem to lead to the conclusion, that the sound cannot be elicited in perfection from spaces bounded by very tense membranous walls, for if we inflate a bladder, or stomach, we shall find that the true tympanitic tone is best obtained when a slight degree of flaccidity is allowed to the parietes. Skoda explains this fact, by supposing that the true drum sound is the result of the vibrations of the contained air alone; and that when the walls are very tightly stretched, the vibrations of the air are interfered with by the vibrations of the walls, by which an imperfect tone is produced. The air in a fully inflated bladder may be considered to be almost in the condition of a rigid body, and we can readily conceive how such a state will interfere to prevent the oscillatory motion of the particles of the included air upon which the production of the sound depends;—that such a view is, to a great degree, correct, is proved by the construction of the ordinary drum, which has always an orifice in some part of its side to allow of the free movements of the internal volume of air. Yet it cannot be denied, that the membrane performs an important part in the formation of the sound, for we know that the pitch of drums depends upon the tension of the drum heads, and that an ordinary drum is capable of producing two distinct notes, in consequence of the two membranes being tuned, by tension, to different notes, usually at the distance of a fourth from each other.

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* Skoda-Abhandlung uber Percuss. und Auscultation, p. 9.

Whatever the rationale of the sound may be, the true tympanitic tone is the usual indication of the presence of air in the cavity of the pleura. This sound is also stated to occur, independently of pneumothorax, in two other conditions of the lung.—First, in the emphysematous portions which frequently surround hepatised lung parenchyma; and, secondly, when the lung is gradually recovering from the compression of fluid previously effused into the pleural space. The explanation of the latter case is not very evident, but having several times observed it, I have no doubt of its truth;—my attention was first drawn to it by Professor Skoda, during my attendance at his cliniques in the Hospital of Vienna.

A modification of the tympanitic tone is called metallic klinging, and is evidently dependent upon the same conditions (more fully developed), as previously mentioned for the production of the drum tone—a large space filled with air, and bounded by good reflecting walls, will produce a klinging or ringing sound upon percussion, just as the same tone can be obtained by striking sharply upon the side of an empty cask.

Bruit du pot fêlé.—The last tone to be mentioned in connection with the subject of percussion, is the "sound of the broken pot." This sound is obtained from superficial cavities bounded by elastic walls, and which also freely communicate with one or more tolerably sized bronchial tubes. Its theory is best explained by reference to the schoolboy's trick of placing the palms of the hands together, and striking the back of one of them against the knee, when the air, being suddenly expelled from the cavity by the force of the blow, rushes out with a hissing sound most strangely called by Laennec, the *bruit du pot fêlé.* The condition, therefore, for the production of this sound in the chest is a superficial cavity of a tolerable size, communicating with the external air by means of one or two bronchi, and surrounded by elastic, compressible walls. The sudden passage of the air of the cavity through some fluid contained in the excavation itself, or the bronchi in communication with it, produces a mixed kind of sound of a gurgling hissing character, very easily conceived. In seeking to produce the bruit, it is often advantageous to direct the patient to open his mouth, so that the force of the stroke upon the chest may more easily expel the air contained in the abnormal cavity.

Piorry, Mailliot, and other writers, have described a great variety of sounds capable of being elicited by the percussion of the thorax, and have exhibited a fund of ingenuity in the application of this means of detecting diseases of all kinds in every region of the body. No organ has escaped their observation; the brain, the kidneys, the ureters, the pancreas, and all the deep-seated viscera, have, according to the statements of these indefatigable experimenters, been made, by the aid of percussion, to declare the lesions to which they may be liable; but for the results obtained, I must refer to the works of these authors. In the above account, and that which immediately follows, I believe that I have collected all that may be deemed of practical value on the subject; and in so doing, I have attempted to simplify the account as much as possible, and have avoided the enumeration even of a number of sounds, the majority of which, I am convinced, exist only in the sanguine imaginations of the observers. Zehetmayer, the late illustrious pupil of Skoda, in reference to this subject, remarks—"The partial predilection with which the enthusiastic champions of plessimetry have blazoned forth the results obtained by its employment, has injured rather than benefited the good cause. Which of us can refrain from sceptical laughter when practical physicians assure us, that percussion has enabled them to discover an hypertrophy of the pancreas; when another detects, during an attack of intermittent, a diminution in the size of the spleen two hours after the first dose of quinine," (in my time, at La Pitié, the period for the production of this phenomenon had been reduced to three minutes;) "when another can decide upon the dilated condition of the ureters, or the presence of calculi in the kidneys; and when percussion is employed for the detection of cerebral diseases, and results obtained stated to be of great importance to diagnosis. Percussion is undoubtedly well able to inform us of the thickness of the skull, but up to the present time, thick and hollow heads have been detected with tolerable certainty, without the necessity of performing a percussion of the cranium."*

Having given the general statement of the theory, I shall now pass to the practice of percussion, and, without entering into a long account of the various kinds of plessimeters, will at once remark, that none can equal the simple and handy one with which we are naturally endowed—I mean the index and middle fingers of the left hand. I have made use of all kinds of hammers for percuting, and a great variety of plates (bone, metal, leather, gutta percha, and membrane,) for receiving the blow, but have abandoned them all for the fingers of the right and left hand, which answer admirably for hammer and plate. This mode is called mediate percussion, but we may occasionally strike with our finger direct upon the chest, a process termed immediate percussion.

The rules for practising percussion are very few, being—

1. To place the patient and yourself in the most convenient position.

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* Grundzüge der Percuss. und Auscult., p. 41.

2. To render the external surface of the chest as firm as possible, by placing the subcutaneous muscles moderately on the stretch. The tension, however, should not be so great that the vibrations of the parts covering the chest should interfere with those produced in the interior of that cavity.
3. To be careful to compare corresponding parts of the two sides of the chest (allowance of course being made for the position of the heart and liver), previous to drawing any conclusions from the examination.
4. To percute with different degrees of force, in order that the deep-seated portions should feel the stroke as well as the more superficial parts of the thoracic organs.

The object of percussion being—

1. To determine the position; and,—

2. To decide upon the density of the viscera contained in the thoracic and abdominal cavities; it is evident that we must at first familiarise ourselves with the condition of the organs in a healthy state, by making ourselves acquainted with their boundary lines, the character of the sounds capable of being elicited from them, and the sense of resistance which they offer to the finger. For the sake of convenience, writers upon the diseases of the chest and abdomen have been in the habit of mapping out the superficies of the body into certain regions, and have usually adopted such arrangements as bear reference more to certain points in the external surface than to the actual position

of the contained viscera. Of the various systems offered, I shall choose the one adopted by my late father, in his 'Lectures upon the Diseases of the Chest,' as being simple, intelligible, and easily remembered.

1. The clavicular region, divided into the sternal, middle, and humeral portions.
2. The anterior superior, extending from the first to the fourth rib.
3. The superior mammary, from the fourth to the eighth rib.
4. The sub-mammary, from the eighth to the twelfth rib.
5. The axillary, from the apex of the axilla to the fourth rib.
6. The superior lateral, from the fourth to the eighth rib.
7. The inferior lateral, from the eighth to the twelfth rib.
8. The supra-spinal.
9. The infra-spinal.
10. The inter-scapular.
11. The dorsal, extending from the angle of the scapula to the last rib.

To the above I would add—

12. The supra-clavicular, as being one of the most important—at least for the purpose of auscultation.

Dr. Sibson has presented another system, which, in distinction to the artificial, above described, may be termed the "natural system." He defines the outlines of his regions by the anatomical boundaries of the subjacent organs. These regions are—

The simple . .	The right pulmonic. The left pulmonic. The cardiac.
The compound .	The pulmo-hepatic. The pulmo-gastric. The pulmo-cardiac (right). The pulmo-cardiac (left). The pulmo-vasal.

These will be best described by sketching the relative position of the lungs, heart, spleen, and stomach,—to the details of which I would wish to direct especial attention.

A vertical line drawn down the middle of the sternum will correspond to the line of contact of the right and left lung, and its lower part will be over the right side of the exposed portion of the heart.

The anterior and lateral boundaries of the right lung are, therefore, respectively represented by this line; and by another, which, commencing at the lower end of the sternum, passes in a direction backwards and to the right side, crossing in its course the cartilage of the seventh rib, the anterior extremity of the eighth, the junction of the anterior with the posterior two thirds of the ninth, and the vertebral extremity of the tenth rib.

The border of the left lung is at first represented by a vertical line down the sternum, as far as the fifth costal articulation; the boundary line then becomes horizontal and parallel to the fifth rib, for a distance of an inch and a half, and then suddenly drops downwards to the seventh rib, where it sweeps backwards and outwards in a manner similar to the inferior border of the right lung, with the difference that the left reaches lower than the right lung. The separation of the two lungs consequently leaves an irregular space, compared by some anatomists to a lozenge in shape; its upper and lower boundaries are the fifth and seventh costal cartilages respectively, and its inner the sternum. The apex of the heart beats between the fifth and sixth ribs, and in some cases between the sixth and seventh. This exposed portion of the pericardium, at the close of an expiration, has an area of two and a half to three square inches, and its longest and shortest diameters are three inches, and two inches and a half respectively.

The right pulmonic region is bounded above by the summit of the right lung, and internally by the mesial line; its base is an imaginary plane resting on the right convexity of the diaphragm, cutting the fifth intercostal space in front, and the articulation of the eighth rib behind.

The left pulmonic region is bounded by the summit of the left lung, and below by an imaginary plane, resting upon the left convexity of the diaphragm, the left side of that muscle being nearly an inch lower than the right side; the left pulmonic will extend that distance further down than the right pulmonic region; the inner boundary is the mesian line, as far as the fifth sterno-costal articulation, where the line follows the curve of the superior border of the heart, which I have already described.

The cardiac region has been already sketched in tracing out the right and left pulmonic.

The pulmo-hepatic is that lamina of lung which caps the summit of the liver on the right side, extending from the fifth intercostal space to the lower border of the chest.

The pulmo-gastric is that portion below the left convexity of the diaphragm, and extending to the margin of the ribs; it caps a portion of the liver, stomach, and spleen.

The pulmo-cardiac, right and left, correspond to those portions whose inner margins cover the right and left sides of the heart respectively.

The pulmo-vasal extends upwards along the sternum, from the origins of the aorta and pulmonary artery opposite the articulations of the third costal cartilages with the sternum. It corresponds to the layer of lung between the sternum and the great vessels formed by the anterior margins of the upper and middle lobes of the right, and the upper lobe of the left lung.

The above arrangement is certainly more scientific than the one previously described, although it must be borne in mind, that the boundaries are constantly varying with the different degrees to which the lung is inflated.

The tone produced by the percussion of the right and left pulmonic regions is clear, full, and resonant, the necessary result of the entire spaces being occupied by lung only.

The cardiac region offers a dull sound over a space considerably modified by respiration, the range of the dullness varying from about eight to three square inches, corresponding to the conditions of extreme expiration and inspiration.

The compound regions give out sounds whose character depends considerably upon the extent to which the lungs are expanded.

Let me now pass rapidly over the general results obtained by the percussion of the healthy chest and abdomen. In the supra-clavicular region the sound is tolerably distinct, being dependent upon the extent to which the apices of the lung rise above the clavicles.

On the right side of the chest the tone is full, clear, non-tympanitic, from the clavicle to the sixth rib, the maximum being obtained in the position of the third rib. Below the margin of the sixth rib, and as far as the lower margin of the chest, the sound becomes gradually duller, and at the lowest part entirely so, unless the individual examined be made to inspire fully.

The upper half of the sternum offers a clear, full tone; the lower half, in consequence of the position of the heart and great vessels, and left lobe of the liver, give out a dull, empty sound, the pulmo-cardiac portions of lung being too thin to produce any considerable amount of resonance.

The left side of the chest is clear and sonorous, from the clavicle to the sixth or seventh rib, with the exception of the cardiac portion lying between the fifth and seventh ribs. Below the point at which the apex of the heart usually strikes, the sound becomes tympanitic, in consequence of the position of the stomach.

The lateral region of the right half of the chest produces a tone which is full and clear, from the axilla to the sixth rib, when it becomes gradually duller towards the lower margin of the chest. The left side gives out a full tone to the line corresponding to the upper boundary of the spleen.

The sounds obtained from the supra- and infra-scapular spaces are dull and fleshy, from the presence of the thick muscular tissue. The inter-scapular region yields a fuller and clearer tone, while below the scapulæ the resonance is very distinct. It must be, however, borne in mind, that from the position of the liver, the clear tone extends lower down posteriorly on the left than on the right side of the chest.

To make the subject complete, I will give the results of the percussion of the upper part of the abdomen. The liver produces a dull and fleshy tone, extending to a distance of two inches to the left of the sternum, three inches from the lower boundary of the chest towards the nipple, and four inches from the same line towards the axilla. We cannot lay down exact rules for the size of this organ in the healthy state; but whenever we find it extending to the fifth rib above or beyond the lower boundary of the chest, we may conclude upon the existence of some morbid condition of the organ itself, or of the parts adjacent. I must remind you that in empyema or hydrothorax of the right side the liver is depressed, in consequence of the increased superincumbent pressure.

The spleen is a body nearly four inches in length, three inches in breadth, and as many in depth. Situated in the left hypochondrium, it lies below the diaphragm, above the descending colon, between the great *cul-de-sac* of the stomach and the false ribs, and in front of the supra-renal capsules and upper part of the right kidney. Its external surface corresponds to the ninth, tenth, and eleventh ribs, and it is separated by the diaphragm from a thin layer of lung. These relations will determine its position. The dull tone obtained by the percussion of the body of the spleen is bounded above by the lung-tone; below, by that of the intestine; and towards the mesian line, by the tympanitic sound of the stomach. Posteriorly, the organ cannot be distinguished from the kidney, as they both rest on the vertebral column.

The position of the stomach must be decided by previously determining the boundaries of the organs which surround it. There is, of course, some difficulty, and oftentimes an impossibility, of distinguishing the stomach-tone from that of the intestine—such as the arch of the colon. To meet this difficulty, M. Maillot very complacently suggests the administration of a quantity of water to the patient!—a means of solving the problem, which, however advantageous to the physician, might perhaps be somewhat objectionable to the invalid.

Having described the limits of the organs in their healthy condition, and the varieties of resonance elicited by their percussion, I shall now proceed to consider the alterations produced by disease. For this purpose, I shall refer to a diagram contained in Zehetmayer's work, as embracing a condensed account of these abnormal deviations.

The air of the cavity of the chest may be—

1. Abnormally increased in quantity.
2. Abnormally diminished in quantity.
3. Entirely absent in certain parts.

1. Abnormally increased.

A. GENERALLY.	*B.* LOCALLY.

A. GENERALLY.

(*a.*) In vesicular emphysema, the sound being clearer and fuller than natural, and extending beyond the ordinary limit of the healthy lung.

(*b.*) In pneumothorax, with or without empyema, the tone is clear, full, and tympanitic, provided the thoracic walls are not too tightly stretched.

B. LOCALLY.

(*a.*) In hollow cavities, a clear, empty sound, often metallic, or associated with the *bruit du pot fêlé.*

(*b.*) In partial emphysema, particularly at the edges of hepatised or tubercular infiltrated lung; the tone is clear, empty, and sometimes tympanitic.

2. Diminished in quantity.

(*a.*) In bronchial catarrh, the sound upon percussion is not altered, unless some alterations in the parenchyma of the lung are coexistent.

(*b.*) In the first and third stages of pneumonia, the dullness of the sound is proportional to the number of lung cells filled with secretion.

(*c.*) In œdema pulmonum, the same.

(*d.*) In small effusions into the pleura, union of the pleura by false membranes, &c., the tone is less full and sonorous.

3. Total absence of air from—
   (*a.*) Deposition of fibrin, tubercular, and encephaloid matter, in the lung-cells and inter-vesicular tissue, (apoplexia pulmonum hepatisation, phthisis, cancer, &c.) In these cases, the sound on percussion is dull to an extent proportional to the quantity and completeness of the lung consolidated.
   (*b.*) Or compression of the general parenchyma in consequence of—
      (*a*) Effusions into the pleural cavity . . { Pleuritis. Hydrothorax.
      (β) Tumours pressing and encroaching upon the lungs—as, aneurism of the aorta; hypertrophy and dilatation of the heart; exudation into the pericardium; morbid (cancerous) masses; ascites; enlargement of the abdominal organs, &c. &c.

The dullness upon percussion in *b*, will be coextensive with the disease; but in none is the perfect absence of all resonance so marked as in the case of the effusion of fluid into the cavity of the pleura.

A very few words will be sufficient for the subject of the resistance presented by the internal organs of the body to the percuting finger. The resistance of a body appears to be inversely proportional to its elasticity—

the greater the elasticity, the less the sense of resistance which it presents. Now, as the air appears to be the principal elastic component in the chest, an abnormal increase or diminution in its quantity will be attended with a corresponding diminution or increase in the sense of resistance—hence, the sense of resistance to the percuting finger will be increased in consolidation and compression of the lung, due to the presence of fluid, and diminished in pneumothorax and emphysema; and it is worthy of mention, that the depression made in the two latter instances in the thoracic parietes by the percuting finger almost instantaneously disappears on the cessation of the percussion. The greatest amount of resistance ever experienced is, in the case of pleuritic effusion, when the fluid drives the lung upwards and backwards towards the vertebral column, expands the side of the chest, and effaces the intercostal spaces.

LECTURE IV.

INSPECTION.—PALPATION.—NORMAL RESPIRATORY MURMUR.

By simply surveying the chest of a patient, we are often enabled to detect, from the outward appearances, the indications of important morbid changes proceeding within its cavity. By inspection we observe the shape of the thorax, the irregularities of form, congenital or acquired, presented by the general surface or portion of its walls; we recognise, at a glance, the characteristic rounded chest of emphysema; the one-sided enlargement due to the presence of fluid in the cavity of the pleura; the contraction resulting from the subsequent absorption of the effusion; the partial sub-clavicular flattening, indicative of the deposition of tubercle or the presence of a cavity; the retraction and collapse of the lower part of the chest during the active stages of croup, hooping-cough, hysteria, emphysema, and other diseases where the diaphragm acts suddenly and violently, and the air does not enter the lungs with sufficient rapidity to occupy the partial vacuum left by the descent of the muscle. By inspection we detect the increased or diminished action of the muscles of respiration, the thoracic or abdominal character of the breathing, its completeness or incompleteness, equality or inequality, and the ease or difficulty with which this important function is performed. We observe the times respectively occupied by the acts of inspiration and expiration, the rhythm of respiration; and we compare the movements of the two sides of the chest in regard to their symmetry, rapidity, and force. Malformations of the chest, abnormal positions of the heart observed in the altered position of the point where that organ strikes the chest wall, dilated and hypertrophied conditions of that viscus, aneurisms of the aorta, &c., are all, within the province of inspection, a means of investigation of evident value in the detection of thoracic disease.

Mensuration.—It is an important fact to be borne in mind, with respect to mensuration, that a perfectly symmetrical chest is of rare occurrence, the ratio of the number of regularly to irregularly formed chests being, according to M. Woillez (quoted by Dr. Walshe) as 41 to 197—a circumstance which is attributable, independently of disease, to peculiarities of occupation, dress, and posture. It must also be remembered, that the right side of the chest is normally half an inch larger than the left, unless the individual be decidedly left-handed, in which case the proportion is often reversed.

Mensuration is employed for two purposes: to determine—

1. The mobility of the chest.

2. The enlargement or contraction consequent upon disease.

For the determination of the mobility, we have only to measure the circumference of the chest during the acts of extreme inspiration and expiration, and the difference between the two numbers expressing those measurements will give the answer required. This mobility or power of expansion varies widely in different individuals, and is, of course, evidently affected by diseases of the thoracic viscera. The mean measurement may be taken, in persons of healthy condition and middle height, at two inches, or perhaps a little more. I have registered in my note-book a few cases which have reached to four, and one instance to five inches. Dr. Hutchinson speaks of an individual whose mobility attained six inches and a quarter, and whose vital capacity exceeded 300 cubic inches. The case to which I allude, of a mobility of five inches, was a gentleman, forty-two years of age, six feet two inches in height, fourteen stone in weight, thirty-seven inches and a half round the chest, and vital capacity 310 cubic inches.

Although the circumference of the upper part of the chest is undoubtedly diminished in the vast majority of cases of phthisis pulmonalis, we have rarely any occasion to make it the subject of measurement, for the stethoscope at this period is alone sufficient to enable us to decide upon the nature of the affection. In ordinary practice we have more commonly to apply this means of investigation to cases of pleuritic effusion, where the diameters of the chest have become considerably altered. The fluid effused may vary in quantity from a few ounces to as much as twenty pints. The mechanical effects of such an accumulation are of course considerable, being a dilatation of the side of the chest more especially affecting its lower portion, obliteration of the intercostal spaces, depression of the diaphragm and of the subjacent abdominal organs, and compression of the lung to one fourth, one sixth, or even one eighth of its normal size. In many instances the diseased side will present an increase of two inches upon the normal measurement. According to Rokitansky, "the contraction which follows is found to affect more particularly the sixth, seventh, and eighth ribs; the space from the axilla downwards becomes scooped and hollowed out; the different diameters of the side are diminished; the ribs sink, and in many cases touch and rest upon each other; the muscles of the side, and more especially the intercostal, waste, become atrophied, and, in proportion to the duration and degree of their paralysis, shrunken, and ultimately converted into fibro-cellular tissue. The vertebral column, in the dorsal region, gradually sinking, curves towards the healthy side, and an opposite curvature taking place in the loins, produces, by the apparent shortening of one leg, the general resemblance of a chronic coxalgia."

Measurement will now exhibit the amount of injury which the chest has suffered in its symmetry and proportions; in some instances a difference of two inches and a half being presented between the healthy and contracted sides. It must, however, be borne in mind, that the healthy side, in consequence of its supplementary work, becomes dilated, and that an allowance must be made on this account in our comparison of the two sides of the chest.

Several instruments have been invented for the purpose of measuring the chest; but I am in the habit of employing a piece of tape, graduated to inches and parts of an inch, as being the simplest and most convenient mode of obtaining the information required.

Palpation.—Several valuable indications of disease can be obtained by laying our hands on a patient's chest, and comparing the distinctness and force of the vibrations felt at different parts of its surface. The vibration of the voice (vocal fremitus) differs considerably in different persons in health—grave voices, *cæteris paribus*, producing the fremitus more distinctly than those which are of a shrill and weak character; hence it is felt less perceptible in women and children than in men. Although the natural vocal vibration is stated by some writers to be more marked on the right than the left side of the chest, the difference, according to my experience, may be practically disregarded, and both sides may be taken to present, in health, the same amount of vocal fremitus. This point is one of considerable importance, as the value of palpation, as a means of diagnosis, entirely rests upon the comparison of the two sides.

I shall not enter, in this place, into the rationale of the increase or decrease of the vocal fremitus in disease, as I shall have to speak more extensively upon the subject, when we arrive at the consideration of bronchophony. The practical results, however, are the following:—The vocal fremitus is diminished with the effusion of fluid into the cavity of the pleura, and is ultimately lost, when the quantity effused is considerable. On the contrary, the vocal fremitus is increased in all cases of consolidated lung, whatever be the cause of that condition (tubercle, ordinary fibrin, plastic lymph, &c.), provided that the larger bronchial tubes which traverse the solid parenchyma, remain open and unobstructed by secretion, so that the vibrations of the voice may be propagated uninterruptedly along the columns of air contained in the tracheal and bronchial passages. I would wish to dwell more particularly upon the latter condition, for I believe that the discrepancy of opinion found among authors, regarding the presence or absence of vocal fremitus, in cases where the lung is undoubtedly infiltrated with solid matter, must be ascribed to a forgetfulness of the state of the bronchial passages which traverse or lead to the consolidated portions. If we bear in mind the fact that the vibrations formed in the larynx are mainly conducted downwards into the chest, along the columns of air contained in the bronchial channels, it will be at once admitted that any break or interruption in these columns by the presence of a quantity of secretion in those passages, will tend to diminish, and ultimately stifle and destroy, all vocal fremitus. We shall find, hereafter, that bronchophony and vocal fremitus are associated in origin, and are affected similarly in the various conditions of the lung. Independently of observation of the vocal fremitus, palpation affords other valuable indications of the presence of certain morbid states of the contents of the thoracic cavity. It enables us to distinguish a displacement or dilated and hypertrophied condition of the heart; to recognise a peculiar thrill, compared by Laennec to the purring of a cat (*fremissement cattaire*), and which is usually indicative of a contracted orifice of the heart; to detect the rubbing of rough exudations upon the pleural surfaces; the fluctuation of fluid when air and fluid coexist in the pleural cavity; and in many cases to feel the vibrations which characterise a narrowed condition of the respiratory air-passages.

Respiratory Murmur.—I have now to direct attention to a consideration of the character and signification of the respiratory murmur—the study of which forms the very basis of the science of auscultation. As the extent of disease occurring in the pulmonary organs is, for the most part, measured by the amount of deviation which the morbid murmur presents from the healthy respiratory standard, we must evidently familiarise ourselves, at the outset, with the character and limits of the normal pulmonary sounds, before attempting the consideration of the alterations which indicate the presence of disease. On placing a stethoscope over the larynx and trachea of an individual, we at once distinguish, during the act of respiration, a loud, dry, hoarse, hollow, rushing sound, evidently resulting from the passage of a current of air over a surface which presents a considerable degree of friction to its progress. This sound, which may be readily imitated by placing the middle of the dorsum of the tongue in apposition with the hard palate, and inspiring or expiring with considerable force, may be traced downwards to the point where the trachea passes into the cavity of the chest, when its tone becomes modified and ultimately changed into one of a totally dissimilar character.

The sound which is audible over the upper bone of the sternum, and very commonly, in thin persons, in the inter-scapular region, at a part corresponding to the root of the lungs, is termed the bronchial murmur, and is well imitated, as suggested by Barth and Roger, by blowing into a roll of paper, or across the orifice of a stethoscope; in its character it bears a close resemblance to the tracheal sound, and only differs from it in being softer in its quality and less loud in its intensity. Considerable importance is to be attached to the determination of the exact limits of the natural bronchial murmur, or tubular breathing, as it is sometimes called. Some diversity of opinion exists among authors upon the extent to which this sound may be heard. M. Fournet limits its seat to the inter-scapular region. Dr. Hughes states that it is sometimes absent in all the localities (the upper part of the sternum, the inner side of the infra-clavicular and the inter-scapular regions); but sometimes, when inaudible on the front of the chest, it may still be heard close to the spine, at a level with the centre of the scapula. Dr. Williams extends it "over the space of from one to two inches on each side of the top of the sternum." My own experience, however, leads me to concur in the view adopted by Dr. Walshe—that while the sound in question is frequently distinguished in the inter-scapular space, its seat in front of the chest is limited to the upper part of the sternum; and that any increase in its extent, right or left of that position, is in general to be regarded as an indication of the presence of some morbid condition of the lung. It must not be supposed because the bronchial murmur is inaudible in health beyond the positions just specified, that no vibrations occur in the air contained in the further bronchial ramifications; on the contrary, we can have no doubt of the presence of these vibrations, and we account for their being inaudible, from two circumstances:—

1. The bronchial ramifications being covered by a layer of lung, whose peculiar vesicular murmur masks the true bronchial sound; and—
2. The lamina of lung, always containing more or less air, presents a non-homogeneous structure, which stifles, rather than conducts, the bronchial vibrations to the chest-walls.

The latter is, perhaps, the more effective cause of the two in rendering the vibrations inaudible.

We have an interesting example of the non-conducting nature of non-homogeneous bodies in the following simple experiment, first suggested by Chladni:—

"If we fill a tall glass—a champagne glass, for instance—half full of that sparkling fluid, it cannot be made to ring by a stroke on its edge as long as the effervescence lasts and the wine is full of air-bubbles, but gives a dead, puffy, disagreeable sound. As the effervescence subsides the tone becomes clearer; and when the liquid is perfectly tranquil, the glass rings as usual; but on re-exciting the bubbles by agitation, the musical tone again disappears. To understand the reason of this, we must consider what passes in the communication of vibrations through the liquid from one side of the glass to the other; and it is clear that if any considerable part of a system be unsusceptible of regular vibration, the whole must be so."—(Herschel, art. "Sound," 'Encyclopædia Metropolitana.')

The third species of sound which is due to the current of air traversing the free passages of the lungs, is the pulmonary, or vesicular murmur—a sound which is soft and expanding in character, and best imitated by gently pronouncing the letter *v* or *f* in a prolonged manner, the air which passes through the orifice between the compressed lips giving a fair resemblance of its tone. The tone is heard most fully in those parts of the chest which contain lung only—the right and left pulmonic regions.

The tracheal, bronchial, and vesicular sounds are each divisible into two periods, corresponding to the times of inspiration and expiration—the tracheal expiratory being nearly as long and as loud as the tracheal inspiratory murmur; the bronchial expiratory is of considerably shorter duration than the inspiratory, while the pulmonary expiratory is at the utmost one fourth the length of the inspiratory murmur, weak in character, and in a great number of instances entirely absent. The relative duration of the inspiratory and expiratory murmurs is deserving of considerable attention, inasmuch as the first indications of disease are generally exhibited by the alterations which occur in their respective lengths, the vesicular expiratory, for instance, becoming either equal in duration and intensity, and in advanced disease longer and louder than the inspiratory murmur. Differences will, of course, be found in the respective duration of the murmurs in different healthy individuals; but it will be sufficient to remember, that in the majority of cases, the times occupied by these sounds are nearly in the ratio of three to one. Thus, if we suppose a healthy individual to breathe fifteen times in a minute, or once in four seconds, the times occupied by the periods of inspiration, expiration, and repose, will be one and a half, a half, and two seconds respectively. Any prolongation of the expiration is made at the expense of the period of rest, which, as disease advances, becomes proportionally shortened, until it is found in many cases to be entirely abolished, when inspiration follows upon expiration, with scarcely any appreciable interval of silence. Having studied the characters of these murmurs, let us briefly consider the causes which produce them, and the conditions which affect their propagation or conduction to the thoracic parietes. And, commencing with the inspiratory murmur, I must, in the first place, remind you that the mass of air drawn into the lungs at each inspiration passes entirely through the trachea, and diverging into the right and left bronchial divisions, becomes subdivided into smaller and smaller streams, until the diameter of the terminal vibrating columns is reduced to one fiftieth of an inch, the diameter of the ultimate bronchial ramifications. A distribution of this kind, which may be compared to the main-pipe and its branches of a system of gas supply in a town, must evidently be so arranged that the air may travel from the main into its divisions, and from any trunk into its branches, with as little impediment as possible. From numerous measurements of the diameters of the bronchial tubes, which remain permanently open during the entire act of respiration, I have found that the sum of the squares of the diameters of the dividing channels is always greater than the square of the diameter of the tube from which they diverge, and that the proportion is maintained in such a manner that the stream of air can suffer but little impediment in the larger tubes from the mode of their division. The further bronchial divisions will not admit of accurate measurement, from their smallness and elasticity; but I think that an observation of the immense number of orifices which lead from them into the proper vesicular tissue of the lung, coupled with a knowledge of the existence of the law in the upper part of the respiratory tract, will enable us to decide upon the probability of a similar relation existing between a small bronchial tube and the ramifications which spring from it. You are aware that the walls of the respiratory tract are lined by a thin, smooth membrane, which is preserved constantly moistened by the presence of a thin exhalation; that the walls under this membrane are composed of cartilaginous rings, which preserve the open condition of these air-channels, but that this character is gradually lost with the progressive subdivision of the bronchi, until the smaller tubes are found to consist of simple membrane. The inequality due to the rings must be one cause of friction, while the firmness of the walls must tend to increase and maintain the resonance of any sounds which may be formed or propagated within them. Another cause of friction is found in the divisions of the tubes, where their edges, presenting themselves in a direction opposed to the current of inspired air, act as vibrating tongues, which tend most materially to render the loudness of the inspiratory considerably greater than that of the expiratory murmur. The effect of the arrangement of these edges may be readily estimated by the simple act of blowing over the leaf and edge of a leaf of thin paper. The sound produced by the current of air impinging upon the flat surface of the leaf is very slight, but is immediately raised in loudness when the air is directed upon the edge; and from this simple illustration we may form an idea of the part which the multitude of bronchial divisions play in the production of the inspiratory murmur. Another, and by no means the least effective impediment to the current of the inspired air, is presented by the natural elasticity or resiliency of the lung—a power which offers considerable resistance to the entrance of atmospheric air, and proportional assistance to its exit from the chest. The nature of this power is seen in the facility with which a portion of lung, which has been stretched, returns to its natural size, and the rapidity of the collapse of healthy lungs upon the equalisation of the atmospheric pressure upon their internal and external surfaces. There is considerable difficulty in estimating the amount which is capable of being exerted by this passive resistance, although the observation of pulmonary disease, and especially of emphysema, leads us to recognise its importance in the mechanism of the respiratory act. To quote the words of Dr. Carson, the ingenious experimenter upon this subject:—"Two powers are concerned in regulating the movements, and in varying the dimensions and form of the chest, the elasticity of the lungs, and the contractile power of the diaphragm; of these powers, one is permanent and equable, the other variable, and exerted at intervals. The contractile power of the diaphragm, when fully excited, is evidently much stronger than its antagonist, the resiliency of the lungs; but the latter not being exhausted, takes advantage of the necessary relaxation of the other, and rebounding like the stone of Sisyphus, recovers its ground, and renews the toil of its more powerful opponent."* To determine the amount of this power, Dr. Carson instituted the following experiment:—an apparatus was constructed, consisting of a glass globe connected with two tubes, of which one was vertical, and the other formed in such a manner as to be capable of being closely fitted into the trachea of any animal, the elastic power of whose lungs was required. The tube was placed into the trachea of a recently-slaughtered bullock; water was then poured into the vertical tube, until it stood in it at a height of more than one foot above the level of the water in the globe; openings were now made in both sides of the chest, without perforating the pleura pulmonalis,—the lungs immediately collapsed, the water rising, and standing two inches higher than before. Now, as the only forces in action, at this time, were the pressure of the column of water and the contractile power of the lungs, it follows that we have, in this manner, a rude measure of the latter force, when circumstances admit of its full play. Similar experiments made upon sheep, calves, and dogs, proved that the elasticity of their lungs was measured by a column of water nearly one foot in height, while in rabbits and cats it varied in pressure from six to ten inches. Dr. Carson having omitted to give us the dimensions of the apparatus employed, we are unable to calculate the exact amount of the pressure exerted in these instances. On referring, however, to Dr. Hutchinson's paper, we find, that in pumping air after death into the chest of an individual, a pressure of one and a half cubic inches of mercury, or nearly twelve ounces avoirdupois, upon every square inch of surface, was required to rupture the pulmonary substance, and we may therefore consider this amount to be the average elastic resilient power of the human lung.

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* Dr. Carson, 'Philosoph. Transact.,' 1820, p. 43.

I have stated the resistances which the current of air must overcome in the perfect expansion of the lung, to be:—

1. The friction of the walls.
2. The friction of the divisions of the bronchi.
3. The resisting elasticity of the pulmonary tissue.

Now, what is the power actually exerted in a full inspiration? We must again refer to Dr. Hutchinson's valuable communication for an answer. An instrument (to which I referred in my First Lecture) was constructed by that gentleman, and intended to measure the comparative forces of inspiration and expiration, in such a way that the forces might be calculated from the respective lengths of the columns of mercury moved during these efforts. By experiment, it was found that the mercury raised by a full inspiration was a column, whose base was one square inch, and height three inches: while in the act of expiration the column was found to be one third higher. Calculating the inspiratory effort, and taking for this purpose the superficies of the internal surface of the chest at 350 square inches, the force exerted is found by the law of the pressure of fluids to be equal to raising a column of mercury whose base is 350 square inches, and height three inches,—*i. e.*,

= 350 × weight of 3 cubic inches of Mercury
= 350 × 1½lb. nearly
= 525 lbs. nearly
= 4¾ cwt. nearly;

i. e., the force exerted by the muscles of inspiration equals the force required to raise four and three-quarter cwts. This power appears enormous; but when we remember the narrowness of the passages through which the air must be ultimately drawn, and the amount of friction which it must encounter in its course, as well as the great resilient elasticity of the lungs, we at once see the necessity for the existence of such a force. And we may place in comparison with this power the calculation made by Dr. Hale, that the left ventricle of the heart acts at every contraction with a force equal to the weight of nearly half a hundred-weight. We may readily test, in a rude manner, the power capable of being exerted by the muscles of inspiration, by attempting, by pressing with the two hands upon the chest of an individual, to prevent his inspiration. We shall find that a pressure, although perhaps amounting to more than 200 pounds, will be compelled to yield to the greater power of the inspiratory muscles. Great as is the inspiratory, we find it even surpassed by the expiratory force. Thus, in a table given by Valentin, ('Lehrbuch der Physiologie des Menschen,' p. 529,) we find that the mean of several experiments made upon himself and others by the pneumatometer, gave the same result as that obtained by Dr. Hutchinson, of the expiratory being one third greater than the inspiratory power. The following is the table given by this distinguished physiologist:—

Individuals.	Age.	Maximum of the Mercurial Pressure in Millimetres.	
		Extreme Inspiration.	Extreme Expiration.
Valentin.	32	130	80
S.	21	232	256
T.	21	220	256
H.	20·5	170	—
I.	20	58	224
J.	18	56	—
Mean	22·085	144·3	204

From the sketch which I have given of the nature of the inspiratory murmur, we have seen that sound, the result of friction, originates in, and is propagated along, the whole course of the respiratory tract—that its character and loudness at any point depend upon the velocity and volume of the air at that part, and that the tracheal, bronchial, and pulmonary murmurs are modifications of, and pass gradually into, each other. It may naturally be objected that the bronchial is distinct in itself, and is not observed to shade off into the vesicular murmur. This consideration brings us to the last circumstance which affects the respiratory murmur; I mean the nature of the pulmonary tissue as a body capable of conducting sound. There can be no doubt that a portion of healthy lung must, like cotton or wool, or any other substance which contains a quantity of air within its meshes or interstices, prove a bad conductor of sound, for any vibrations originating within it will have to pass from membrane to air, and from air to membrane, many hundred times before reaching the surface of the chest. I have already shown the non-conducting power of a non-homogeneous substance in the case of an effervescing fluid, stifling the vibrations of a glass containing it; and I think that the consideration of what I said at the commencement of my remarks upon the respiratory murmur will be sufficient to account for the presence of the vesicular murmur alone in every part of the chest, with the exception of those positions where the bronchi are close to the thoracic parietes,—viz., under the upper bone of the sternum and in the interscapular region.

Expiratory Murmur.—We have seen that the act of ordinary expiration is due to the relaxation of the diaphragm, the elasticity of the ribs and cartilage, and the resilient power residing in the pulmonary substance. By the combination of these forces, the air is expelled from the lung cells into the smaller bronchial tubes, and driven thence into the large channels; columns of air press onwards to the outlet with increased force, velocity, and volume, until they ultimately converge in one large tube—the trachea. As the stream of air which issues from the lung cells and terminal bronchi is small in quantity and velocity, the vibrations produced will be also weak, and conveyed imperfectly, or not at all, to the surface of the chest. But as the columns converge, effect junctions, and increase in volume and velocity, there will be a corresponding increase in the resulting sound, until, in the general gathering in the primary bronchi, the quantity of air collected, and the force of its vibrations, will be sufficient to produce a distinct murmur to the ear. This is the true natural bronchial expiratory murmur. The act of expiration is performed in one third the time occupied by that of the inspiration—a result evidently depending upon the greater force of the expiratory powers, and the fewer obstacles which exist to the exit of the air from the interior of the thoracic cavity.

LECTURE V.

RESPIRATORY MURMUR.—BRONCHOPHONY.

In my last Lecture I entered rather fully into the consideration of the Respiratory Murmurs, under the conviction that an intimate acquaintance with the nature and causes of these sounds in the healthy state is absolutely essential to the comprehension of the alterations which they exhibit in disease—alterations which depend upon conditions affecting the diameters of the air-passages, the resiliency of the lung parenchyma, and its capability of conducting sound. Although subjects of a controversial character should be avoided as much as possible in Lectures which simply aim at teaching the elements of a science, I feel that I should be omitting important matter, were I not to allude to certain theories which have been advanced by various authors, in explanation of the respiratory sounds, and which, however much they may appear to me to be unsatisfactory, deserve, on account of their propounders, some degree of consideration and attention. I will, at first, advert to the views entertained by Dr. Blakiston, respecting the causes of the vesicular murmur, as contained in a valuable work published by that gentleman on certain diseases of the thoracic viscera. Dr. Blakiston commences by denying that the lung vesicles offer any resistance to the entrance of atmospheric air, considering that "the lung-cells are similarly situated to a bladder placed under the bell of an air-pump, which communicates with the external air through the neck. If this communication be for a moment interrupted, by turning a stop-cock in the neck of the bladder, and the pump be worked then as the air around the bladder becomes rarefied, that within it becomes proportionally rarefied, expands, and exerts an equable outward pressure against the internal surface of the bladder, with little or no friction: the stop-cock being then turned, the external air rushes in. This is just what takes place during inspiration, under the influence of the muscles, which elevate the ribs, and enlarge the capacity of the thorax, except that there is no appreciable interval between the expansion of the air already in the vesicles, and the entrance of additional air in the bronchial tubes. No greater resistance, therefore, is offered to the entrance of air into the vesicles than during its exit from them, and, consequently, no more sound can be engendered in them during inspiration than expiration. Hence it follows, that the respiratory sound, which is more intense during inspiration than expiration, is not principally formed in the air vesicles."

To account for the presence of the inspiratory murmur, Dr. Blakiston supposes the existence of muscular fibres around the smallest bronchial tubes, which, by contracting during inspiration, for the purpose of driving the air with greater velocity into the lung-cells, form obstructions to the current. The obstructions thus formed produce a greater velocity, and, consequently, an increased friction of the inspired air, and therefore a louder pulmonary inspiratory sound. During the act of expiration, the air, by the relaxation of these muscles, is allowed to escape without impediment, and, consequently, with scarcely any appreciable sound. The seat of the pulmonary sound is, therefore, according to Dr. Blakiston, the smaller bronchial tubes; and its proximate cause, the contraction of muscular fibres supposed to encircle them. Ingenious as this theory may appear, it will be found to be untenable, for the following reasons:—

1. The lungs undoubtedly exert a decided power of resistance to the entrance of atmospheric air, as is proved by their rapid collapse after inflation, by their continued expansion in emphysema, where their elasticity has been impaired, and by the results already mentioned of Dr. Carson's experiments.

2. Dr. Blakiston virtually admits the existence of this resistance, in assigning a muscular function to fibres arranged around the minute bronchial tubes, for the increased velocity and force of the inspired air, consequent upon their contraction, can be only intended to overcome some resistance existing in the lung-cells.

3. As an objection, which ought to have preceded the other two, we have the authority of the best microscopic observers, Messrs. Quekett and Rainey, for asserting that the terminal bronchial tubes and intercellular passages present no traces of any kind of circular muscular fibre whatever.

Again, in explanation of the vesicular murmur, M. Emile Beau advanced a theory that the respiratory murmur was nothing more than the resonance in the columns of inspired and expired air of the "bruit guttural," *i. e.*, the sound resulting from the air impinging upon the velum palati and neighbouring parts. This view was found to be untenable when it was shown that the destruction of these parts by syphilis, or any other disease, in no way affected the pulmonary murmur. The same physiologist further extended his view by making the glottis the source of the sounds audible over the general surface of the chest; an explanation found to be equally groundless on considering that—

1. The respiratory murmur bore no relation, in its intensity, to the loudness of the sound produced in the glottis.

2. In the whoop of pertussis, the vesicular murmur is entirely abolished, while the sound at the glottis is exceedingly loud.

3. Ulceration of the glottis has no effect in modifying the pulmonary sound.

4. The inspiratory murmur has its usual normal character in cases where the respiration is carried on through an artificial opening in the trachea.

Such are the views entertained by these distinguished writers respecting the origin of the true vesicular murmur; and I shall conclude this subject by simply recapitulating the more important facts at which we have arrived. These are:—that the acts of inspiration and expiration producing friction of air against the parietes of the air-channels in every part, are attended by sound in every portion of those passages; that the sound heard at any given point of the chest is partly the result of vibrations originally formed at that part, and partly of vibrations propagated to it; that the tracheal and bronchial murmurs are the loudest, because the tubes in which they are produced are the largest in diameter, possess the firmest walls, and are placed in the most superficial position; that the pulmonary murmur is soft and diffuse in its character, by reason of the smallness of the cavities in which it is produced, and the non-homogeneous character of the medium which it is mostly compelled to traverse to reach the chest-wall, and that its intensity is directly proportional to the force with which the inspiration is effected, and to the amount of the resistance which the column of air has to encounter; that any cause which affects the smoothness of the lining membrane, the diameters of the air-passages, or the elasticity of their parietes, will modify the character of the sounds formed in those channels; and lastly, that any change in the density of the pulmonary substance around the tubes and cells, will affect the conduction of the sounds to the thoracic parietes in a degree proportional to the alteration.

Bronchophony.—Having thus discussed the nature and theory of the healthy respiratory sounds, it would be naturally expected that I should proceed at once to the changes which those murmurs undergo in a diseased condition of the pulmonary organs; but I think it more advisable to commence with the auscultation of the voice, as an easy introduction to that part of our subject. On examining the anatomical construction of the larynx, we perceive that two distinct folds of mucous membrane, which, diverging from the angle of the thyroid cartilage, project horizontally from the lateral walls, and pass respectively backwards to be attached to the bases of the arytænoid cartilages. These folds are termed the inferior or true vocal chords, and, inclosing a quantity of yellow fibrous tissue, form the thyro-arytænoid ligaments. Above them are situated two similar folds of mucous membrane, which are, however, deficient in the peculiar tissue just mentioned, and have, consequently, been styled the false vocal chords, from a supposition of their playing an unimportant part in the production of the voice. Between the inferior vocal chords is situated the rima glottidis, an opening triangular in shape, having its apex in the thyroid, and its base formed by the anterior portions of the arytænoid cartilages. All physiologists agree that the inferior vocal chords are the principal sources of the voice; that the differences in pitch depend upon the degree and mode in which the five pairs of muscles, which regulate the larynx, contract or dilate the glottis; and that the entire apparatus partakes of the conjoined characters of a reed and stringed instrument. The small vibrating chords rarely exceed an inch in length, while the range of the notes which they are capable of producing is usually two octaves, and in some remarkable singers considerably greater. My object in entering into these details, which must be familiar to the majority present, is to draw attention more particularly to the mode in which loudness of tone is secured, as the principles involved bear at once upon the theory of normal and abnormal bronchophony. It must, in the first place, be evident that the vocal chords owe a great proportion of the intensity of the sound which they are capable of emitting to the peculiarity of their position, for strings similar to them in thickness, length, and elasticity, would be unable to produce tones of such loudness if made to vibrate in the open air without the advantage of a sounding chest. A tuning-fork in vibration is heard but faintly until placed in contact with a table or sounding-board; and a jew's-harp, however forcibly its tongue may be struck, produces but little sound until placed in front of the mouth—an instance, proving that the particles of the air contained in the cavity of the mouth are thrown into vibrations, which combine with the original tone, and increase its loudness. The sounding-chest of a violin or guitar are familiar examples of the same principle, which is, however, best exemplified in a peculiar instrument of the Japanese, called the gender, which was first introduced into this country by Sir Stamford Raffles. "This instrument consists essentially of metallic plates, suspended by two strings in a horizontal position; beneath each plate is fixed an upright bamboo, of a proper length, to reciprocate the sound of the plate. When the plates are struck, a rich and full tone is produced by the consonance of the columns of air contained in the respective tubes." The vibrations of the sounding-board, of the air in the chest of the violin, and in the bamboo pipes of the gender, are termed consonating vibrations, and the original sounds are said to be increased by consonance. Consonance is regulated by fixed and definite laws. Thus, if we hold the vibrating extremity of a tuning-fork over the orifice of an empty wine bottle, a slight increase of sound will be obtained; but by pouring water into the bottle to a point to be found by trial, we shall discover a length of column of air, whose reciprocal vibrations will increase the intensity of the original sound to a remarkable degree. From an extensive series of experiments, Mr Wheatstone ('Quarterly Journal,' vol. iii,) has deduced the law, that a column of air may vibrate with consonance when the number of its vibrations is any multiple of those of the original sounding body. "Thus, when a tube closed at one end was furnished with a moveable piston, Mr. Wheatstone found that the tone of a tuning-fork was reciprocated by a column of air six inches in length. The column was then diminished to three inches, and the octave of the original sound was produced. By using forks of lower tone, and very small tubes, and adjusting the length of the column of air, he obtained the octave, twelfth, double octave, and other concords of the original sound. The same experimentalist found that one column of air may be made to consonate with another; thus, let two flutes, which are in unison, be placed near each other; let a certain note be produced on one flute, and the intensity of the sound will be increased by the consonating vibrations of the air contained in the second flute in a proportion dependent upon the distance between the two instruments." ('Philosophy of Sound,' by Higgins.) The instances which I have adduced will be sufficient to indicate the nature of consonance, and the laws which regulate its production. The conditions requisite for consonance are found to be present in the larynx and trachea, viz.:—

1. A cavity of considerable size.
2. Walls of sufficient smoothness to reflect any waves of sound produced within them.

And as these cavities are simply subcutaneous, the voice which is heard in them, upon the application of a stethoscope, is found to be sharp and penetrating, and often unpleasantly acute to the ear. At the division of the bronchi, not only has a diminution taken place in the caliber of the air-tubes, but the passages have become surrounded by the spongy tissue of the lung, and enclosed also within the bony cavity of the chest. Hence the natural bronchophony is conveyed very indistinctly to the ear, and is heard only in those positions where the bronchi are nearest to the surface,—viz., the upper bone of the sternum and the inter-scapular region over the second and third dorsal vertebræ.

"On the entrance of the bronchi into the pulmonary tissue the tubes no longer possess cartilaginous rings, but contain small irregularly-shaped plates of cartilage imbedded in fibrous tissue. These plates become thinner, less frequent, and smaller, with the progressive ramifications of the bronchial tubes, until the smallest passages consist of thin membrane only. In the normal condition of the lung parenchyma, the voice consonates much less strongly in these passages than in the trachea, and its weakness is proportional to the smallness of the quantity of cartilage contained in the walls of the tubes." (Skoda.)

In fact, all trace of the voice is lost over the general surface of the chest, with the exception of the positions already mentioned. In disease, however, an increased resonance of the voice may be found to take place at almost any part of the chest, and with a distinctness and clearness, varying from the weakest bronchophony to a resonance, which may surpass, in intensity, the natural voice of the patient heard at the larynx. To the most marked form writers have given the name of pectoriloquy, while the terms bronchophony and tubular breathing have been assigned to the less voluminous species. A strict line of demarcation cannot be drawn between them, for their difference will be found to exist in degree only. The conditions requisite for their production are—a cavity of a certain size, which must be comparatively empty; be formed by firm walls, capable of reflecting sound; have a direct communication with the larynx, and be surrounded by a medium capable of conducting sound to the chest-wall. Whatever be the nature of the cavity—whether a large bronchial tube or artificial space hollowed out in the substance of the lung—the conditions for the production of bronchophony or pectoriloquy will be the same, and the intensity of the sound will depend upon—

1. The size of the tube or cavity.
2. Its uni- or multi-locular form.
3. The smoothness and elasticity of its walls.
4. The nature, size, and freedom from secretion of the communications between the cavity or tube and the larynx.
5. The quantity of consolidated or compressed lung around it.
6. The proximity of the tube or cavity to the chest-walls.
7. The presence or absence of fluid in the interior.

The distinctness and loudness of the sound will be found to depend upon the number of the above conditions which are found to coexist. The theory which I have adopted in explanation of bronchophony, is entirely based upon the views of Professor Skoda, of Vienna, and only differs from it in ascribing a greater conducting power to solid than to spongy lung. In corroboration of his views, this author advances the following experiments, which, although of extreme simplicity, admirably illustrate the theory. He begins by stating that the bronchial tubes, after death, generally contain a quantity of mucus, blood, serum, &c., which interrupts the communication between the deeper tubes or excavations and the larynx, and renders all experiments upon the lung difficult and uncertain in result. The modifications in the character of the voice in normal and diseased lung may, however, be studied in an easier manner.

"The membrane of the small intestine will very fairly represent the more membranous portion of the bronchial tubes, in its capability of reflecting sound, while the liver, or heart, will correspond to consolidated lung. Take an inflated portion of intestine, and having placed two stethoscopes upon it, at some distance from each other, speak through one of them. A person at the other instrument will hear the voice consonating in the intestine. If a piece of liver, lung, or intestine, filled with water, be placed between the intestine and the stethoscope, the consonance of the voice will be found to be diminished, and to be very weak, or entirely abolished, when the substance interposed is only half an inch in thickness, and merely covers the mouth of the stethoscope. If we dip the inflated intestine under water, and repeat the experiment, taking care to exclude the fluid from the stethoscopes, it will be found that the consonance of the voice in the air contained in the intestine is much louder than when the experiment was made out of the water. If a passage be bored in a liver, without penetrating to the opposite side, and this be spoken into by means of a tube accurately fitting the opening, the voice will be heard through a stethoscope placed along any part of the whole length of the passage, and for a considerable distance on each side of it, with a strength and intensity much surpassing the voice of the speaker which is heard through the free air. The same can be heard through several inches thick of liver or lung substance, bone or cartilage, with an intensity inversely proportional to the thickness of the interposed stratum. If the liver be dipped under water, taking care that the fluid does not enter the passage which has been bored in it, the voice may be heard, by means of the stethoscope, through a stratum of two or more inches of water. This experiment is more easily performed with the heart than the liver. Empty the left side of that organ of its contents, tie up the auricular opening, and having destroyed the aortic valves, speak through a tube introduced through the aorta into the left ventricle. The consonating vibrations of the voice can be heard through a stethoscope placed upon the heart, and also through layers of lung or liver substance, or through a stratum of water.

"These experiments, in my opinion, show how the degrees of the intensity of the voice, in the thorax, are related to the different conditions of the lung. If the voice in the intestine, when out of the water, consonated so feebly as to be inaudible through a half-inch layer of lung, liver, or water, the consonance of the voice in the membranous bronchial tubes will likewise be so slight as to be almost, if not entirely, inaudible at the parietes of the chest. But, on the contrary, as the voice, in the passage through the liver, and in the ventricle of the heart, consonated so strongly as to be distinguished through several inches thick of interposed substance, so will the voice in the bronchial tubes of a hepatised portion of lung, or within the excavations of a tubercularly-infiltrated lung, consonate so powerfully as to appear louder in the thorax than that which issues from the mouth into the free air."*

The theory usually adopted by writers in this country is the following:—The waves of sound, which descend from the larynx into the bronchial tubes, become gradually diminished in intensity with the progressive division of the bronchi; and whatever amount of vocal vibration exists in any of the smaller passages is prevented from reaching the thoracic parietes, by having to traverse a quantity of healthy air-containing lung. Supposing, however, that the pulmonary substance around the tubes becomes consolidated, homogeneous, and consequently a better conductor of sound, the vocal vibrations will be transmitted, with facility, to the chest-wall, and become audible over the consolidated lung.

This view is, I believe, correct, to its fullest extent; but I cannot consider it sufficient to explain the entire phenomenon of morbid bronchophony. Simple conduction can never cause the abnormal bronchophony to appear louder than the voice of the patient, heard at the larynx, or issuing from the mouth; nor can it account for the disappearance of the sound, and its reappearance after the patient has freely expectorated.

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* Skoda—Abhandlung über Percussion und Auscultation, p. 46.

And again: if homogeneity be a condition of good conduction, how does it happen that "bronchophony cannot be regarded as a sign of pleurisy, with effusion—at the stage of effusion, with dilatation of the chest, all vocal resonance has ceased." (Dr. Walshe, p. 257.)

On the other hand, consonance is a principle admitted by writers on acoustics; and it is an indubitable fact, that a column or mass of air, enclosed within smooth channels, will reciprocate to a sound produced by a vibrating plate, cord, or another column of air.

We see that the bronchial tubes, or artificial cavities, surrounded by air-less lung, present conditions favorable for reciprocation, and afford an easy explanation, by the doctrine of consonance, of the remarkable intensity of the voice frequently observed in certain diseased conditions of the pulmonary organs.

"Ought we not, on physical grounds, to suppose that the voice would resound more powerfully, the denser and more uniform the sound-conducting lung parenchyma, the nearer it is situated to the thin and elastic chest-walls, and the more sonorous and powerful the voice which sounds within it?"*

Whatever theory be adopted, all writers agree in the main fact, that the lung-parenchyma must be rendered air-less, either by deposit of solid matter in the vesicles, or by strong pressure from without, to produce bronchophony; and as the morbid conditions which produce such structural alterations may exist, in every degree, from the slightest to the most complete obliteration of the air-spaces, so will the resonance of the voice vary, from the slightest increase of its tone to the degree of loudness which I have more than once described.

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* Zehetmayer, Grundzüge der Percuss. und Auscult., p. 84.

Although the boundary between strong and weak bronchophony must be necessarily arbitrary and uncertain, it will be found useful, in practice, to distinguish between a bronchophony which is attended with a feeling of vibration to the ear of the auscultator —"the voice which," according to Laennec, "completely penetrates the stethoscope"—and a sound, similar in character, but unattended with this delicate fremitus of the walls of the chest. Both conditions may accompany the same morbid state of the lung; both may be, and are, usually associated with bronchial respiration; and both attended with dullness on percussion.

The pathological condition indicated by a loud and intense bronchophony, is, in the great majority of instances, an extensive consolidation of the pulmonary substance with or without an abnormal enlargement of the channels or spaces in which the air circulates. The substance producing consolidation may be fibrin (apoplexia pulmonum), inflammatory lymph (pneumonia), tubercular, or cancerous matter, &c.;—the abnormal spaces may be vomicæ, resulting from pneumonic abscesses or cavities formed by the softening of tubercular deposit. An obliteration of the vesicular texture is also found around simple bronchial dilatations, and, according to Rokitansky, is, to a great extent, to be considered as the cause of the peculiar enlargements of those tubes—a view which is decidedly at variance with the doctrine taught by Laennec. This great authority states, that "the second form, *i. e.*, the sacky dilatation of the bronchi, is not developed in the catarrhal portion of the bronchial system, but beyond it, and is the consequence of inflammation of the terminal branches of the bronchial tubes; of obturation by the secretion poured out, swelling of the mucous membrane, and ultimate true obliteration. The parenchyma belonging to the part of the obliterated bronchial system falls together, and produces a vacuum which is filled out by the dilatation of the tube. The collapse of the lung at length terminates in complete obliteration, whereby the parenchyma shrinks into itself, produces a greater vacuum, and consequently greater bronchial dilatation;"* a view, which, he adds, approximates this theory to that of Dr. Corrigan's. The intensity of the resonance of the voice in this condition of the lung will depend upon the same conditions as I have already described, as regulating the bronchophony of other morbid conditions of the pulmonary substance. Its character will in no way point out the peculiarity of the space in which it is produced; the true nature of which must be determined from the position in the chest where it is best heard, from the results derived from percussion, and from the concomitant symptoms and general history of the patient. Weak bronchophony, unaccompanied by the sensation of vibration, may attend the various morbid conditions above described, and is also frequently present, under certain conditions, when a large quantity of fluid exists in the cavity of the pleura.

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* Rokitansky, Handbuch der Pathol. Anatomie, vol. iii, p. 9.

The immediate effect of a considerable amount of effused fluid in the pleural space, is the compression of the lung towards the vertebral column, and the production of bronchophony in the interscapular region. Unless adhesions already exist capable of binding some portions of the lung to the lateral and anterior walls of the thorax, all traces of the voice will be lost over the general surface of the chest, with the exception of the position just indicated. The presence of old bands of false membrane attaching the lung to the costal surfaces, will lead to the formation of bronchophony at these parts of the chest, and will often puzzle the auscultator. In such cases we must depend upon the results given by percussion, the observation of the displacements of the heart and other organs, the measurements of the chest, &c.

Setting aside the cause of these adhesions, we have, in the existence of loud bronchophony, a valuable diagnostic sign between pleuritic effusion and pneumonic, or tubercular consolidation,—in the former (if the sound occurs at all), the vocal resonance is weak, and its loudness in no degree proportional to the dullness evinced by the percussion of the diseased side, while in the latter instances, the bronchial voice is usually excessively marked, and the dullness on percussion limited only to the space corresponding to the consolidated lung. And, as a further diagnostic mark, you may bear in mind that while the vocal fremitus is diminished in pleuritic effusion, it is abnormally increased in hepatised or tubercularly-infiltrated lung.

I have only to add to this sketch of Bronchophony and its cause, that I have found the sound to be best developed by making the patient whisper, in place of speaking or counting aloud; the slower and longer waves thus formed appearing to find their way more readily down the bronchial channels, and to establish with greater facility consonating waves in the passages which traverse the air-less lung parenchyma.

LECTURE VI.

ÆGOPHONY.

This sound is so closely allied to Bronchophony, that I shall take the opportunity of speaking of it in this place. It consists of a peculiar resonance of the voice which accompanies or follows the words of the patient. The voice appears thin, clear, superficial, and trembling in its tone, and derives its name, Ægophony, from the resemblance which it bears to the bleating of a goat. Sometimes it appears like Punch's voice, and hence the French writers have called it *la voix de Polichinelle*. Its usual position is at the lower and posterior part of the chest in the proximity of the larger bronchi; it is frequently found distinctly limited to a small space near the angle of the scapula, but it may be occasionally traced round the chest, encircling it like a girdle. The sound is so characteristic, that, once heard, it can never be forgotten. Its theory has been the subject of considerable discussion among writers on the diseases of the chest. Laennec asserted that ægophony never exists without the presence of fluid in the cavity of the pleura, that the sound characteristically marks the existence of a very thin layer of the fluid, and that it is simply the resonance of the voice in one or more of the larger bronchial tubes, which have become flattened by the pressure of the effusion. He believed that the voice in its descent into the bronchial ramifications becomes altered in a peculiar manner upon entering the flattened tubes, and obtains its trembling character from having to traverse the thin edge of the fluid. According to this author, therefore, three conditions are requisite for the production of the sound:—

(*a.*) Flattened bronchial tubes.

(*b.*) Compressed lung.

(*c.*) Slight effusion of fluid into the cavity of the pleura.

His reasons for the theory were derived from observation, experiment, and analogy.

1. The position of the sound at the lower angle of the scapula, between that point and the spine, proved that it occurs where the bronchi are large and numerous, and where, when the effusion is smallest, the layer of fluid is the thinnest. The shifting of the sound, with an alteration in the position of the patient, its gradual disappearance with an increase in the quantity effused, conjoined with the fact of its reappearance with the reabsorption of the fluid, proved that fluid existed in the pleura, and that its presence in a thin layer was essential to the production of ægophony.

2. Laennec placed a bladder, half-filled with water, on the interscapular region of an individual whose natural bronchophony was excessively marked, and he

stated that the voice heard through it was thin and trembling in its character.

3. From analogy, Laennec conceived the end of a flattened bronchus to represent the mouth-piece of an oboe or clarionet—a reed instrument. "The reed is a narrow aperture, in which an elastic plate, called a tongue, is so placed that when excited by a current of air it may, by its vibrations, alternately close and open the aperture. Hence the reed is intended to produce a continued and periodical interruption in the continuity of the stream, the pitch of the note being regulated by the number of interruptions made in a given time." To obtain a full, rich sound, it is necessary that the air in a tube attached should consonate with the note produced by the reed. This is the simple theory of all reed instruments; and Laennec imagined that the bronchial tubes flattened at their ends represented a wind instrument of this kind. The explanation, however, only served to account for the occurrence of ægophony in the larger bronchial tubes near the angle of the scapula, to which spot the sound would be limited if a flattened condition of these channels constituted the sole cause of the sound. As ægophony was found to extend, in some instances, around the chest, Laennec further supposed that the layer of effused fluid assisted in forming the trembling, bleating tone, and promoted its conduction along the walls of the thorax. And, indeed, in some cases, the small quantity of fluid effused appeared to be the most important element in the production of the sound. For in convalescence from pleuritic effusion, the contraction of the side, and consequent constant pressure

upon the bronchial tubes, should be attended with permanent ægophony, supposing Laennec's original view to be correct—a result, however, not found to accord with bed-side observations.

The objections to the above theory are—

1. That ægophony has been found to be present in cases of simple consolidation of the lung, where no effusion of fluid has been detected in the cavity of the pleura. Thus, Barth and Roger, who follow in Laennec's wake, "admit the existence of cases where a certain degree of ægophony is perceived in the absence of fluid in the pleural sac; and, of other instances, where the sound is wanting, although the presence of liquid, even to a moderate extent, has been capable of being demonstrated. It has been observed to disappear in some patients, after having lasted for some time, without any change having taken place in the level of the effusion, and without any reason being readily assigned for its disappearance."*

And, again, with regard to the thinness of the layer of fluid requisite to its production, Dr. Walshe states, "although the rule is, with respect to ægophony, that it diminishes and disappears with the increase of effusion, yet cases do occur in which it remains, in spite of very abundant accumulation. I have seen such cases, and such a one has been published by Andral, where displacement of the diaphragm and heart gave evidence of the abundance of the fluid."

2. That the voice, heard over the interscapular

Barth et Roger, 'Traité Pratique d'Auscultation,' p. 201.

region of women and children in perfect health, has often a silvery, trembling tone.

3. That, even granting the occurrence of this peculiar flattening of the bronchial tubes, no real analogy can be traced between such a condition and the free vibrating tongue of a reed instrument; and I may add,—

4. That from frequent repetitions of the experiment of the bladder, pressed over the larynx and trachea of a healthy individual, I have never been able to obtain any modification of the natural tracheophony comparable to ægophony.

Skoda informs us, that he has detected ægophony as well when fluid was present in the pleura as when it was absent—that he has found it in pneumonia, and in cases where the parenchyma was infiltrated with tubercular matter; and he adds, "If it is true that Laennec's ægophony may exist without the presence of fluid in the cavity of the thorax, we may naturally suppose that Laennec's mode of explaining its origin by the vibration of a thin layer of fluid is not correct, at least for all cases." Supposing, then, that Laennec's view be considered unsatisfactory, what theory can we find to replace it? How can we account for this remarkable modification of the voice, and what are the conditions essentially requisite to its production?

Skoda denies that any alteration in the position of the patient affects the character, or causes the disappearance, of the ægophonic voice; and he maintains, that a trembling interrupted tone can proceed only from the impulse of one solid body upon another, or upon fluid, or aeriform bodies. Thus, a tuning-fork will produce a trembling tone, when its vibrating extremity is placed in the lightest contact possible with another body; and every one is acquainted with the effect of the impulse of a column of air upon thin paper capable of vibration. I allude, of course, to the child's trick of making music with a comb. Have we any condition in the lung which may be considered analogous? Skoda believes ægophony to be a form of bronchophony, caused by the reaction of the walls of a bronchus traversing consolidated lung, upon the consonating vibrations produced within it—a view which, in my mind, is as inexplicable as Laennec's; but he adds, that a portion of mucus, &c., which imperfectly closes the mouth of a bronchial tube, may, by its vibrations, produce the sound in question.

Fully admitting the difficulty of explaining the phenomenon, I am inclined to the belief, that tenacious secretion of some kind answers the purpose of a vibrating tongue in the bronchial tube, or tubes, in which the sound is produced, and that ægophony is, consequently, a species of bronchophony, accidentally modified by this circumstance. We may, therefore, infer consolidation or compression of the pulmonary substance to be the conditions indicated by this sound; and we may add, that a slight amount of effusion of fluid in the pleura, while it favours the consolidation of the lung, is also capable of diffusing the ægophony, in the manner in which it has been stated to be occasionally found. In adopting this view of the origin of this peculiar sound, I am aware that I am advocating a theory which is opposed to the majority of writers upon the subject, in this country; but I do so from a belief that the arguments adduced in favour of Laennec's ingenious explanation are insufficient to establish its correctness, and from having personally examined many cases, where, although ægophony was present, I could not assure myself, by other means, of the existence of fluid in the cavity of the pleura.

In conclusion, I would only add one remark to this subject: that if such careful observers as Barth and Roger, in France, and Dr. Walshe, in this country, admit the possibility of the occurrence of ægophony, in simple pneumonia, without pleurisy, we are driven to the inference that the sound is not a characteristic mark of the presence of fluid in the cavity of the pleura, as laid down by Laennec. Its meaning must be interpreted, to a great extent, by the other co-existent signs obtained from the examination of the chest.

Abnormal Alterations of the Respiratory Murmur.—In a preceding part of this course, I pointed out the existence of two distinct sounds in the normal condition of the respiratory organs which are easily distinguishable from each other, and limited to distinct regions—the pulmonary or vesicular murmur, audible over the greater part of the surface of the chest, and the bronchial, which is confined to the upper bone of the sternum and interscapular region. I shall now describe the numerous modifications which these respiratory sounds undergo in disease, and shall commence with the abnormal alterations in the vesicular murmur.

The anomalies of the vesicular murmur are divisible into those of—

1. *Intensity*
    - Increased.
    - Diminished.
    - Absent.
2. *Rhythm.*
3. *Quality.*

1. *Intensity.*—The pulmonary sound being the direct result of the friction of the inspired air against the walls and divisions of the smallest bronchial tubes and air-cells, it will follow that when a greater number of vesicles than usual are expanded, or when the cells usually in action are distended to a greater amount than ordinary, the increased volume of the air inspired, and the greater force and rapidity of its transit, will be attended by an increase in the loudness of the respiratory murmur. This increased intensity of the murmur due to increased pulmonary action, is termed, when occurring in adults, puerile respiration, from its supposed resemblance to the loud tone of the respiration in children, and supplementary, from its supplying an amount of respiration which is deficient in some portion of the pulmonary organs. This increased murmur is therefore, in itself, a normal condition, the intensity alone being altered, while the natural relation between the respective loudness and duration of the inspiratory and expiratory sounds remains unchanged.

Effusions of fluid in one of the pleural cavities, tubercular or inflammatory deposit in one side of the chest, produce puerile respiration to a greater or less extent in the other side, and its presence should always lead us to search with attention for some cause which is evidently arresting, in some degree, the action of some portion of the lungs. I need scarcely remind you of the necessity of thoroughly investigating the entire chest, and of avoiding the error of mistaking a naturally loud pulmonary murmur for a local increase of that sound. A careful comparison of both sides will ensure the impossibility of committing a mistake of this kind.

Diminished Pulmonary Murmur.—The causes capable of leading to this result are very numerous, and may be classed as follows:—

- (*a.*) Abnormally formed chests from—
    - (α.) Disease of the bony structures, rachitis, &c.
    - (β.) Pleuritis, producing alterations in the form and size of the thoracic cavity.
- (*b.*) Deficient muscular action, from—
    - (α.) General debility, chlorosis, &c.
    - (β.) Local pain
        - Rheumatism of respiratory muscles.
        - Acute stage of pleurisy.
        - Peritonitis impeding the action of the diaphragm, &c.
- (*c.*) Internal obstructions to the free passage of air, from—
    - (α.) Spasmodic closure of the glottis, pertussis, asthma, &c.
    - (β.) Foreign bodies lodged in the trachea and bronchi.

- (γ.) Increased thickness of the bronchial mucous membrane.
- (δ.) Secretions of all kinds in the tubes and cells.
- (ε.) Tubercular, fibrinous, cancerous, and other deposits in the interior of the air-passages.

(*d.*) External compressions.

- (*a.*) Upon the trachea and bronchia
  - Aneurism of the aorta.
  - Bronchocele.
  - Enlarged bronchial glands.
- (β.) Upon the lung
  - Effusions in the cavity of the pleura and pericardium.
  - Enlargement of the heart.
  - Tumours, thoracic and abdominal, dropsy, &c.

(*e.*) Deficient pulmonary elasticity.

Vesicular emphysema.

You will observe, therefore, that the causes which are capable of producing a diminution in the intensity and distinctness of the respiratory murmur are very numerous, and require some degree of experience in their discrimination. I shall draw your attention more particularly to those diseases which are important from their frequency, and marked, in some of their stages at least, by a characteristic weakness of the respiratory murmur. These diseases are—

1. Tubercular deposit, especially the disseminated form.
2. Moderate effusions into the cavity of the chest.
3. Vesicular emphysema.

The commencement of tubercular disease of the lung is mostly indicated by the comparative absence of the respiratory murmur, the diminution of its intensity depending upon the partial compression and obliteration of the air-cells, the obturation, more or less, of the smaller bronchial tubes which lead to the vesicles, and the impediment to the expansion of the diseased portion of the lung consequent upon the local pleurisy so frequently attendant upon this morbid deposit. A deficiency of the respiratory sound at the apex of one lung coexistent with puerile vesicular murmur at the apex of the other is at all times to be considered a suspicious condition; and when associated with prolonged expiration of the suspected apex, dullness on percussion, and an increase in the vocal fremitus of the same part, almost sufficient to establish the certainty of the existence of tubercular disease.

A certain amount of fluid effused into the cavity of the pleura will, by compression of the lung, obliterate the superficial layer of its cells, and removing the entire organs from the walls of the chest, produce a proportionate diminution in the intensity of the respiratory murmur. The diminution of the sound is of course most marked towards the base of the chest, where the fluid is mostly accumulated. The vocal fremitus is diminished, and the sound elicited by percussion, dull in proportion to the quantity of effusion present in the cavity of the pleura.

I have explained, in a former Lecture, the mode in which the inspiratory act is opposed, and the expiratory assisted, by the natural elasticity or resiliency of the lung; and I have referred to Dr. Carson's experiments, in proof of the power which the normal lung is capable of exerting in the expulsion of the air from the air-cells. Bearing this view in mind, it will be readily allowed, that any disease which causes an over-distension of the walls of the lung-vesicles must impair their elastic or resilient power, and thereby diminish their capability of aiding in the act of expiration. From this condition, it is evident that a small quantity of air only can be expelled from an emphysematous lung, and an equally small quantity admitted at the next act of inspiration, and that with this comparative local stagnation of respiration must be associated a diminution in the intensity of the respiratory murmur. The resilient power which still remains acts slower, and consequently produces a prolongation of the expiratory murmur.

Whatever be the immediate cause of emphysema, whether natural atrophy or fatty degeneration of the pulmonary membrane of the cell-walls, as stated by Mr. Rainey, ('Medico-Chirurgical Transactions,')—whether the result of increased functional activity due to a prolonged state of puerile respiration, or the simple result of mechanical dilatation produced by the impediment offered to the exit of the air in the vesicles through the obstructed bronchial passages, the contractility of the diseased portion of the lung is decidedly impaired, and its capability of assisting in the act of expiration partially abolished. And when we add, that in an emphysematous lung partial obliteration occurs of the capillary system which ramifies under the cell-walls, we adduce another reason why the admission of the air can be but of little avail for the purpose of oxygenating the blood, and why the inspiratory murmur should in this disease be expected to suffer a diminution in its intensity. Hence, weak inspiratory murmur, prolonged expiration, loudness on percussion, and diminished vocal fremitus, characterise a permanently over-distended condition of the pulmonary cell-walls. Bronchitis is, in the majority of cases, antecedent to, or coexistent with, vesicular emphysema, and will, of course, present symptoms superadded to those already mentioned.

Total Absence of the Respiratory Murmur.—The causes previously enumerated as being capable of producing diminished respiratory murmur may, when exaggerated, lead to a total absence of that sound; but in ordinary practice we shall find this symptom to be, in the greater proportion of cases, the result of the exudation of a large quantity of fluid in the cavity of the pleura. A mass of tubercles may so obliterate a portion of lung as to render it impermeable to the entrance of air; an emphysematous condition of the vesicular walls may exist to such an extent as to paralyse their respiratory powers; an obstruction may lodge in a bronchial tube, and by completely filling its interior, entirely abolish the respiration of the corresponding portion of the lung; and other causes might be cited as capable of leading to the same result; but the perfect absence of all vesicular murmur is, in the vast number of instances, to be referred to collections of fluid matter in the pleural cavity. The minimum quantity requisite to produce this effect is at present undetermined. The amount effused, in many cases, sometimes reaches to ten, fifteen, or even twenty pounds —a collection of fluid which is evidently sufficient to compress the entire organ against the vertebral column, and to prevent the formation of a vesicular murmur in any portion of its structure. A condition of this kind is easily recognised by this state of the respiration, the total dullness of the sound obtained by percuting the diseased side, the absence of vocal fremitus, the signs afforded by mensuration, and the observation of the position of the heart and great vessels.

2. *Rhythm.*—By this term is meant the relative duration of the periods of time occupied by the acts of inspiration and expiration. The movements of respiration may be accelerated, and in place of being, in the adult, from fifteen to eighteen in a minute, may reach to thirty, forty, or even sixty, in the same time, or may diminish in frequency, and sink as low as eight or ten. In such cases, the inspiratory and expiratory acts usually maintain the same relation in point of time, and the rhythm is, in consequence, preserved unaltered. In the normal condition of the pulmonary organs, the expiratory follows immediately upon the inspiratory murmur, and a small interval of repose occurs, previous to the commencement of the next inspiratory act. In disease, however, the sound upon expiration may be increased in duration and intensity, and may equal, and even surpass, the inspiratory murmur in these qualities. The prolongation of the expiration is a symptom of the highest importance, and deserving of our most careful

attention. As a general rule, we shall find it to be indicative of two conditions, which may exist single or combined; these are—

1. An obstruction to the passage of the air through the bronchial tubes.

2. An impaired condition of the elastic power of the parenchyma of the lung.

The physical alterations capable of obstructing the bronchial channels are very numerous, and will engage our attention more particularly when we arrive at the subject of the various râles audible in the thorax. I shall, in this place, merely speak of the deposition of tubercle in the structure of the lung, and I would strongly impress upon your minds the fact, that a prolongation of the expiratory murmur is the earliest indication of the invasion of this terrible disease. Tubercular matter presents itself in the lungs under two forms, the interstitial and the infiltrated tubercle; the one deposited external to the lung-cells, in the tissue between the smallest lobules and the vesicles, in the shape of roundish, grey, semi-transparent (miliary) little bodies, which form, by their pressure upon the walls, a corresponding prominence in the cavities of the cells and air-channels, and ultimately lead to a complete obliteration of the vesicular spaces; the other, contrary to the interstitial variety, being deposited within the interior of the air-spaces, upon the free surface of the membranes lining these cavities; and producing, according to Rokitansky, a hepatisation of the lung, or rather, a hepatised state, consequent upon tubercular deposit. Both varieties must evidently injure the elastic or resilient power of the lung, and by their projections into the air-channels, impede the exit of air from the vesicles, which are still capable of being inflated during inspiration. Hence both forms will be attended by a prolonged and somewhat rude expiratory murmur. "At the same time that the presence of a certain number of tubercles at a point in the lung diminishes the intensity of the inspiratory murmur at that spot, another phenomenon is found capable of being produced—a louder murmur than usual during the time of expiration. This is accompanied by a blowing much more marked than that which coincides with the movement of inspiration."*

I have already explained the nature of vesicular emphysema, and the condition of the parenchyma of the lung—the same diminution of elastic power which weakens the inspiratory murmur will produce a prolongation of the expiratory sound. Bronchitis is also the most constant accompaniment of emphysema, and hence two causes are found in co-operation, to lengthen the duration of the expiratory murmur—viz., the obstruction resulting from the thickened and swollen condition of the bronchial mucous lining, and the impaired resiliency of the structure of the pulmonary membrane. The last form of altered rhythm is presented in the interrupted respiration, the "respiration saccadée" of Laennec. In place of an almost continuous passage of the inspiration into the expiration, the former act is performed, as it were, by instalments, with the intervention of minute intervals of repose. Timidity, nervousness during the act of examination, the pain from pleurisy or pleurodynia, the physical effect of false membrane, which prevent the rapid expansion of the lung, spasmodic asthma, &c., frequently produce an irregularity in the act of respiration, of the interrupted kind. M. Barth adds, that the "respiration saccadée" is often connected with those tubercular affections which are so frequently accompanied by partial pleurisies of the summit of the lung. The suspicion of a lesion of this kind will be confirmed, if the respiration is rough at the same time that it is interrupted, and if the phenomenon is limited to the superior part of the lung;—it will be changed almost into a certainty if there is, at the same time, dullness upon percussion, and a depression at the corresponding point of the thoracic parietes. On the whole, however, the symptom is of but little practical value, and undeserving of further consideration.

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* Andral, 'Clinique Médicale.'

LECTURE VII.

BRONCHIAL RESPIRATION.

At our last meeting I commenced the consideration of the abnormal alterations of the Respiratory Murmur. I stated that the anomalies of that sound were divisible into those of Intensity, Rhythm, and Quality. I entered rather fully into an account of the deviations in the intensity and rhythm, and have now to describe the changes in the quality or character, of the pulmonary sound. When speaking of the normal respiratory sounds, I described the character of the bronchial murmur, and stated its position to be restricted to the upper bone of the sternum, and to the interscapular region. In disease, this sound may be found to occur in any portion of the chest, and to present every degree of intensity, from a weak, imperfect, and indeterminate tone, to a loud, distinct, and penetrating murmur, which is audible during inspiration and expiration, and, in the greater number of instances, of louder intensity during the latter than the former period. From the character of its tone, it has obtained the names of tubular breathing, bronchial souffle, and blowing respiration. Its position may be deeply seated within the pulmonary parenchyma, or superficial, and apparently immediately beneath the thoracic parietes. From the post-mortem examination of patients who have presented, just previous to death, well-marked bronchial respiration, we find the following conditions to be constantly present, and absolutely essential for the production of the sound:—

1. A bronchial tube, or space of certain size.
2. Consolidated or compressed lung,—*i. e.*, airless parenchyma surrounding the tube or space.
3. Absence of secretion in the tube or space, so that a perfect freedom of communication may exist between the cavity and the upper part of the respiratory passages.

A portion of lung thus circumstanced will be evidently entirely useless for the purposes of respiration, and we should naturally expect a diminution in the muscular action of that portion of the chest-wall which is situated over the diseased pulmonary structure. Dr. Sibson's valuable paper, before referred to, supplies us with a corroboration of the view. In a note, he mentions the following as an instance:—"Dr. Barlow favoured me with the examination of a female affected with pneumonia of the lower lobe of the right lung. The movement over the diaphragmatic ribs of the affected side was ·1 inch; over the healthy side, ·3 inch. The abdominal parietes, which had but little motion below the left tenth rib, actually fell in from ·06 to ·1 inch over the corresponding point of the right side. This case is an additional proof that non-motion, or even reversed motion of the diaphragm on the affected side, is often an indication of pneumonia affecting the base of the lung." In many instances I have been able to verify this statement. And I believe we may most safely conclude that the air circulates but slowly in a bronchus or space, which is surrounded by a mass of consolidated or compressed lung, and that the opinion entertained by Andral, of the bronchial murmur being caused by the inspired air impinging with greater than the normal force upon the walls of a bronchial tube situated as above described, rests upon an entirely erroneous supposition. The more common explanation, which considers bronchial respiration to be simply the ordinary sound normally occurring in the bronchi, and abnormally conducted to the thoracic walls by reason of the better conducting power of consolidated and compressed lung parenchyma, is, in my opinion, insufficient to account for the great intensity of the sound occasionally found in cases of pneumonia and tubercular consolidation. The bronchial breathing is often as distinct as if the patient were blowing direct into the stethoscope, and in many cases as loud as the tracheal murmur itself, and this, in a tube which must be considerably smaller than the trachea, and situated at some distance from the chest-wall. This loudness of tone cannot be the result of simple conduction; on the contrary, some of the vibrations must be lost in their passage through the lung-substance, however homogeneous it may have become, and all of them be weakened by the time they have reached the parietes of the chest. Again, it is impossible to imagine that the normal pulmonary murmur should be able to drown and entirely mask a sound which, as I have already stated, often equals the tracheal and laryngeal respiration in intensity, especially when we remember that the normal expiration is almost inaudible, and therefore incapable of producing such a result.

I would rather adopt the view of Professor Skoda who explains the existence of abnormal bronchial respiration upon the principles of the doctrine of consonance, which I have already described when speaking of bronchophony; but I would admit, what this author is unwilling to allow, that the sound is transmitted with greater facility through consolidated than through healthy air-containing lung. Zehetmayer gives the following brief account of this author's theory:—"The vibrations which produce murmurs in the larynx, trachea, and bronchi, are necessarily propagated to a column of air, which stagnates in a bronchial tube, surrounded by consolidated lung parenchyma. This column is thrown into similar vibrations, the sound produced in the upper part of the air-passages being found to consonate in it with the same strength with which it is perceived at its seat of origin. While now, the respiratory sound is heard merely as a weak murmur in the neighbouring healthy part, (the normal lung parenchyma being, on account of its spongy structure, a bad conductor of sounds originating within it,) the sound which extends from the larynx and trachea, becoming concentrated in the blind-ending bronchus, is still further increased by reflexion from the firm walls, and finding a good conductor in the solid lung-substance, reaches the ear of the auscultator with the same intensity as the murmur at the larynx. Bronchial respiration is therefore the consonance of the respiratory sounds of the larynx and trachea in the column of air contained in a bronchus, which traverses a portion of consolidated lung-parenchyma." As the natural bronchial expiratory is louder than the natural bronchial inspiratory murmur, so the abnormal expiratory will be louder than the abnormal inspiratory murmur. Consolidation being the chief condition essential to the existence of bronchial respiration, the sound will be found to be present in the second stage of pneumonia, (principally found at the base and posterior part of the lung,) in tubercular solidification of the apices, in bronchial dilatations surrounded by airless parenchyma, in consolidation which results from the coagulation of simple fibrin, (apoplexia pulmonum,) and, in fact, wherever a considerable portion of the cells and smaller bronchi has become blocked up by any kind of solid matter whatever. It must be added, however, that a patch of consolidated lung may be so centrally situated, and so covered by a stratum of healthy lung, as to present no stethoscopic sign, and even no symptom, upon percussion. Although somewhat out of place, I may mention, with respect to the occasional fallacy of percussion, or rather its failure to detect consolidated lung, that an interesting case is recorded by Dr. Graves, in his Clinical Lectures, where an extensive tubercular consolidation of the apices was attended by a full, clear sound upon percussion, the portions of healthy lung between and above the tubercular masses being sufficient to counteract the dullness resulting from the diseased pulmonary structure.

In concluding the account of bronchial murmur, I have a few remarks to make respecting its occurrence in cases of effusion into the cavity of the pleura—a subject upon which some difference of opinion exists among writers on auscultation. In twenty-six cases of pleuritis attended with effusion, noted by MM. Barth and Roger, nine only presented any traces of bronchial respiration, while the remaining seventeen exhibited no symptom of it in the deepest inspiration, and in two cases only of the nine was the sound sufficiently strong and distinct to merit the name of bronchial souffle; in the majority of the instances, the sound was loudest during expiration, and in two cases the murmur was entirely expiratory. Dr. Walshe considers true bronchial souffle to be of rare occurrence in pleuritis, and that while the respiration may have a slight bronchial character when the effusion is laminar, no trace of it will be found when the fluid has collected to a considerable extent in the pleural cavity, except at a spot close to the spine, and under the clavicle, where the murmur will be found to be harsh, bronchial, and even sometimes blowing. My own experience entirely accords with this view respecting the rarity of bronchial respiration in this form of disease, and I think that a little consideration will readily show the probability of such a result. A small quantity of effusion will be insufficient to produce any considerable amount of compression, and will be, therefore, incapable of condensing the lung, and placing it in a condition favorable to the production of consonating vibrations. A large amount of effusion will drive the lung upwards and backwards towards the vertebral column, and by removing it from the general parietes of the thorax, render any sounds produced in the respiratory tract incapable of reaching the anterior and lateral walls of the chest. In either case, the bronchial respiration (if existing) must be weak in its character, and its intensity utterly disproportioned to the extreme dullness of the sound obtained from the percussion of the diseased side. In my late father's work, no mention is made of bronchial respiration as a sign of pleuritic effusion; on the contrary, it is stated, that "as soon as a thin layer of fluid is formed between the pleuræ, the respiration almost entirely ceases;"* and in another passage—"as the fluid increases considerably, the lung becomes so compressed that very little air enters into it, and the respiration is entirely lost." "There is, however, a certain point in the chest at which the inspiratory murmur is almost always present during this stage; it is between the base of the scapula and the vertebral column—a space which corresponds to that occupied by the compressed lung." We may therefore conclude, that pleuritis attended with effusion or a collection of liquid occurring from any cause between the pleura costalis and pleura pulmonalis is recognised, among other symptoms, by absence of distinct bronchial respiration over every portion of the chest, with the exception of the interscapular, and, in some cases, subclavicular regions. Such is the general rule with respect to the accumulation of fluid in the pleural space. There exists, however, an important class of exceptions, with which you should be made acquainted; I mean, those cases where adhesions between the pleuræ bind the same parts of the lung firmly to the side of the chest, and prevent its being fully pressed back towards the vertebral column. An extensive laminar adhesion of the upper portion of a lung will, in consequence of the accumulated fluid in the lower part of the chest, lead to the compression of that organ against the spine and upper part of the thoracic wall, and produce a distinct bronchial respiration and bronchophony over the portions of the chest which correspond to the parts compressed. In a case of this kind, noted by Dr. Williams, the sounds were so audible as at first to induce a belief in the existence of a cavity in the upper part of a chest. In allusion to adhesions which maintain the upper part of the lung in close proximity with the chest-wall, while the remainder of the organ is compressed and driven upwards and backwards by the accumulated fluid, Dr. Williams says, the lung "in this condition may still admit air; but as its vesicular structure is much compressed, the sound of respiration will be tubular or bronchial, and a noisy bronchophony will be transmitted by it to the whole upper region of that side. I have often heard the voice and respiration quite tracheal from this cause, and I have been more than once deceived by it into the belief that there were caverns underneath."* A statement of this nature, and emanating from so great an authority in all matters relating to the thoracic viscera, is highly important, and serves to prove, to a great extent, the statement which I advanced in a former lecture, that the sounds proceeding from a normal bronchial tube, surrounded by consolidated or densely-compressed lung, are often identical with those obtained from an abnormal cavity in the pulmonary parenchyma; or, in other words, that no difference of kind exists between bronchophony and pectoriloquy or bronchial and cavernous respiration; for, I repeat, we have, in the quotation above given, a proof that a tube traversing consolidated lung presented to an experienced auscultator the signs of an abnormal space hollowed out in the parenchyma of the lung. Reverting to the subject of the adhesions, we find that the lung may be connected by them to the chest in every variety of way, and that bronchial respiration may be discovered in every part of the thoracic parietes. A long strip of lung may traverse a collection of fluid, in consequence of its intimate attachment to the chest-wall, and by the compression which it suffers from the fluid surrounding it, become so condensed as to be placed in a condition most favorable for bronchial respiration and for increased vocal resonance. Cases of this kind are always difficult to decipher, and require considerable experience for the discrimination of their real nature.

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* 'Lectures on Diseases of the Lungs and Heart,' by Dr. Thomas Davies.

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* Dr. C. J. Williams's 'Lectures on the Physiology and Diseases of the Chest.'

Râles; Rhonchi.—I have already stated, that in the normal condition of the respiratory organs, the mucous membrane which lines the air-passages is smooth, even, and maintained in a constantly moistened condition by a thin watery exhalation bedewing its surface. The physical alterations to which the air-passages are liable may be stated in the most general terms to be the following:—

1. A diminution of calibre depending upon—
    (*a.*) External compression.
    (*b.*) Increased thickness of their walls, due to inflammatory, tubercular, or other diseases.

2. An accumulation, to a greater or less extent, of fluid matter—mucus, pus, blood, serum, &c.

In the greater number of instances, diminution of diameter and increased secretion are found to coexist,—both causes produce the common effect of impeding the entrance and exit of the atmospheric air;

and both lead to peculiar modifications in the character of the respiratory sounds. The alterations thus produced are divisible into two grand varieties, depending for their distinction upon the circumstance, whether the respired column of air is compelled to traverse passages which are simply contracted in their diameters, or whether with or without this contracted condition the column has to force its way through a collection of accumulated secretion. The first class includes what are termed the *Dry sounds*, respectively named—

(α.) Rough respiration.
(β.) Rhonchus sonorus.
(γ.) Rhonchus sibilans.

The second class are the moist sounds, and the result of bubbles of air, of various sizes, bursting in the air-passage, after their escape from the fluid matter through which they have traversed.

Rough Respiration.—The dry condition of the bronchial mucous lining, which characterises the early invasion of bronchitis; the congested condition of the vessels beneath the pulmonary vesicular membrane due to any cause, active, passive, or mechanical; the presence of a thin layer of tough, tenacious mucus in the course of the respiratory tract; the deposition of miliary tubercle, either on the free surface of the mucous structure, or in the intervesicular tissue of the lung, being causes productive of a diminished condition of the caliber of the air tubes, will evidently increase the sources of friction to the respired air, and, consequently, give a certain character of roughness to the murmurs which accompany the acts of inspiration and expiration. This roughness may be limited in extent, or generally diffused, persistent for a time, to give place to the natural murmur, or terminate by passing either into the sonorous and sibilous râles, or into true bronchial respiration. Whatever may be the cause to which this condition of the respiratory murmur is due, we always find it associated with a prolongation of the expiratory murmur, a condition which, as I have already explained, depends upon the difficulty experienced by the expired air in making its way through the narrowed bronchial channels. The transition of rough respiration into rhonchus sonorus or sibilus is readily detected, but the passage into weak bronchial respiration is not always recognised with equal facility. We must seek, in the results afforded by percussion, palpation, and in the concomitant symptoms, the means of establishing the diagnosis. Lastly, with regard to this sound, I would wish you to be fully impressed with the practical fact, that persistent rough respiration, limited to the apex of one lung, and attended by a prolongation of the expiratory murmur, is one of the earliest and most valuable indications of the presence of tubercular deposit in the pulmonary structure.

Rhonchus Sonorus; Rhonchus Sibilans.—A column of air traversing a contracted channel will be thrown into vibrations and produce a sound, which will be grave or acute, loud or weak, according to the size of the orifice through which the air has to make its way, and the force with which it sweeps along the passage.

The narrower the chink the more acute and whistling will be the character of the tone produced. It is evident, therefore, that a contracted condition of the larger bronchial tubes may present every variety of sound, from the deep note of a large orifice to the whistle of a narrow chink; or in other words, that rhonchus sonorus and rhonchus sibilans may be produced in the upper portion of the respiratory tract. On the other hand, any swelling of the membrane which lines the smaller order of bronchi, or any exudation which equally obstinately obstructs their passage, must necessarily contract the channel to so small a size, as to render every sound occurring in these tubes sibilant in its character. Rhonchus sibilans, therefore, while it is not excluded from the larger, is a characteristic mark of the presence of obstructions in the smaller divisons of the bronchial system. The two sounds, therefore, differ only in degree, and are the results of similar morbid conditions occurring in different portions of the respiratory tract. The grave or sonorous râle has generally been compared in its character to the snoring of a sleeper, or to the tone emitted by the base string of a violoncello. The sibilant râle, as its name indicates, resembles somewhat a whistle; by Laennec it was compared to the cooing of a turtle-dove; in fact, the mixture of sounds proceeding from the chest of an old asthmatic patient presents an indescribable medley of noises, which no language can well describe — snoring, piping, whistling, cooing, chirping, and grunting varieties striving for the mastery, and being not only audible to the patient, but to those who may be at some distance from him. The sounds may be heard sometimes during inspiration, sometimes during expiration only — often in both periods; their intensity is frequently so great as to communicate a sensation of vibration to the hand placed upon the parietes of the chest. Occasionally they may persist for a long time at a particular spot, or suddenly vanish from that position to reappear at another,—a result usually following the act of violent expectoration,—they may be confined to one portion of the chest, or, as is more commonly the case, diffused over the generality of its surface, pure and unmingled with any other sound or associated with crepitation of the larger kind, (subcrepitant râle.) The sibilant is usually more persistent than the sonorous variety, and less capable of being removed by coughing or crepitation; it also masks the vesicular murmur much more completely, and is, on the whole, indicative of a greater lesion of the respiratory functions, and associated with a more intense degree of dyspnœa.

Both rhonchi may (though the result is rare) disappear without leaving any other sound beyond those which are natural to the lung, but in the greater number of cases they will be found to make way for the appearance of the moist râles; in fact, the presence of secretion appears to be a very common cause of the grave variety especially, for, as before stated, that sound is found to vanish and reappear, after coughing and expectoration, in a manner which can only be explained by attributing its origin to the presence of some tenacious secretion, which is sufficiently tough to refuse the passage of air through it, but which may, by the full sweep of a column of expired air, be driven along the course of the respiratory tract, until it is entirely expelled from the lungs. The diseases indicated by these rhonchi are acute and chronic bronchitis, with or without emphysema; the coexistence of the latter affection with an obstructed state of the bronchial channels evidently tending to prolong, to a greater extent, the expiratory murmur. Tumours pressing upon the bronchi, and approximating their internal surfaces, and foreign bodies lodged within the bronchial channels, will, I need scarcely repeat, afford the conditions requisite for the production of these rhonchi. Lastly, as the deposition of tubercular matter is always attended by symptoms of inflammation of the smaller bronchial tubes implicated in the disease, be careful to bear in mind that bronchitis, permanently present at one or other apex of the lungs, affords a very strong suspicion of the commencement of consumption.

Moist Sounds result from a portion of the respired air being made to traverse a quantity of fluid collected in a bronchial tube or cavity; the bubbles of the air which emerge from the fluid break upon its surface, and produce a crackling, or, to use the more classical term, crepitant sound. Dr. Williams gives the following very philosophical description of a bubble: "A bubble is a portion of air contained, and slightly compressed, by a thin film of fluid, which preserves its continuity by its molecular or aggregative attraction; when this attraction is overcome by the gravitation of the liquid, the motion of the air, or any other disturbing cause, the bubble bursts; as it bursts, the air from it, slightly expanding, gives to the adjacent air an impulse which, if forcible enough, produces sound." The elements which determine the size of the bubble and the compression to which its contained air is subject, are—

1. The diameter of the space in which the fluid is collected.
2. The tenacity of the fluid.
3. The force of the respiratory act.

Hence it follows, that while bubbles of all sizes may be produced in the larger bronchial tubes and morbid cavities, the lung vesicles and minute bronchial divisions can only admit of bubbles which are excessively small, and nearly equal in size. The different varieties of crepitation, arranged in accordance with this view, are,—

1. Rhonchus crepitans;
2. Rhonchus subcrepitans; mucosus of some writers;
3. Gurgling;

which have a common origin, and differ from each other simply in the size and number of the bubbles producing them.

LECTURE VIII.

RHONCHUS CREPITANS—CREPITATION.

The character of this important sign has been compared to the noise made by the rubbing of dry hair together—to the bubbling made by an effervescing fluid—to the sharp, distinct crackle of salt thrown upon fire—and to the sound resulting from the compression of healthy lung with the fingers. The character of its tone is best understood by the word crackle, the bubbles producing it being evidently very nearly equal in size, very minute, and formed in the smallest divisions of the air-passages. At the same time, it must be admitted, that the sound is not so sharply defined as to be always capable of being accurately distinguished from the subcrepitant râle. Crepitation occurs almost exclusively during inspiration, and is always confined to its point of origin. It is neither removed by cough nor expectoration, but is generally found to be persistent for some period of time. According to the opinion of the majority of writers upon auscultation, rhonchus crepitans is supposed to originate in the mode already described—viz., from the passage of air during inspiration through a quantity of fluid contained in the lung cells and terminal bronchial divisions; and in accordance with this view, Fournet has termed it an intravesicular râle. This author has subdivided this sound into further varieties, corresponding to pulmonary congestion, pneumonia, œdema, and acute catarrh, under the belief that the moistness of the sounds is sufficiently distinct in each case to establish a diagnostic mark between them,—an excess of refinement which you will find to be by no means borne out in practice. In fact, I believe that nothing has tended more to discourage the student in the study of auscultation than the immense variety of sounds which he finds laid down in books, as occurring in pulmonary disease, and which he must be supposed to be able to detect before he can be allowed to be a master of the science. The excessive subdivision of sounds has only served to throw a mystery around the whole art of thoracic examination, and to deter the student from pursuing a subject which is really very simple in itself, and requires a very slight acquaintance with the laws of acoustics to comprehend. Reverting, however, to the theory of crepitation, I must inform you that Dr. Walshe has advanced an explanation of the production of this sound which is at variance with the one usually adopted, and which I cannot pass over in silence, proceeding, as it does, from so great an authority upon the physical diagnosis of the chest. This writer believes the cause to reside in an interstitial plastic exudation, which glues the parenchyma of the lung together, and the dry crackling sound to proceed from the rapid and abrupt unfolding of this glutinous mass during the act of inspiration. The objections which may be offered to this view are—

1. Ordinary pneumonia (the most common cause of crepitus) is not marked by the exudation of a gluti-

nous mass into the extra-vesicular tissue. The viscous character of the sputa in this disease would, on the contrary, lead us to the opinion, that a secretion of such a nature takes place into the lung-cells rather than into the areolar tissue between them. Again, according to Rokitansky,[*] the usual seat of interstitial pneumonia is the apex of the lung, in the interstitial areolar tissue; and he adds the important fact, that common croupal pneumonia is usually associated with that form of disease. I may add, that interstitial inflammation of the lung differs very widely in its characters from those of common pneumonia. An albuminous deposit is thrown into the pulmonary areolar tissue surrounding the vesicles, and becomes ultimately converted into a tough, fibro-cellular substance, in which the cells are embedded, and often obliterated. The portions of lung thus affected sink in, collapse, and draw the surrounding parenchyma into the form of a cicatrix,—a process which may occur at the apex of the lung without any proof existing of the presence of the slightest amount of tubercular deposit. I would wish you to bear this important pathological point in mind, as also the fact mentioned by the same great authority, that the white, tough streaks, which are often found ramifying through the pulmonary substance, result from true interstitial pneumonia.

2. Supposing the opinion to be correct, of a glutinous exudation surrounding the lung-cells, it is not self-evident to my mind how the expansion of such a mass would be attended with a crackling noise, especi-

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[*] Handbuch der Path. Anatomie, vol. iii, p. 108.

ally when we bear in mind the probability of secretion of some kind being present in the lung-cells.

3. True rhonchus crepitans is not confined to pneumonia; it is met with in the early stages of pulmonary œdema, when simple serum is commencing to be exuded from the vessels which ramify under the pulmonary membrane, forming the vesicles.

In fact, wherever the sound exists, no proof can be given that the vesicles are free from fluid secretion. On the contrary, the presence of secretion is indubitably proved by the course of the case; even these instances of interstitial pneumonia, which I have described being associated, according to Rokitansky, with the common form of inflammation of the lung, cannot be made to support the view of the râle being due to secretion peculiarly extravesicular. That the various kinds of crepitation are really allied in character, and that true crepitus in no way differs from the rest in the mode of its origin, is confirmed by MM. Barth and Roger, who state that the subcrepitant "se confond avec le crepitant" and is distinguished from it, not so much by any distinctive characters of sound as by its occurrence during expiration as well as inspiration; and we have the further evidence of Skoda, Zehetmayer, and the majority of our English writers, for the truth of the statement, that the two sounds (one of which is admitted by Dr. Walshe to be due to the passage of air through fluid) shade gradually into each other, and, in fact, appear to be modifications of one another. With reference to the objection urged against the ordinary theory of the sound in the absence of true crepitation during the act of expiration, I can easily imagine that the air, having forced its way through fluid into the interior of the lung-cells, may be expelled from those little cavities, without being compelled to pass a second time through the secretion. At the end of an inspiration, the cells are fully expanded, and space is afforded for the presence of air and fluid. At the commencement of the expiration, the more elastic fluid—the air—is first driven out, finding a free passage from the vesicles into the terminal bronchial ramifications. I think, therefore, from the evidence adduced, we may safely assert that Laennec's rhonchus crepitans, or vesicular crepitation, is the direct result of the passage of air through secreted fluid, and that any attempt to establish the nature of the exudation in the lung-cells (whether mucus, pus, serum, or blood,) is a refinement upon which no real reliance can be placed.

Rhonchus Subcrepitans.—This sound is admitted by all authors to be due to the bubbling of air through fluid. Its character, however, entirely depends upon the size of the bubbles, and the number formed at every act of inspiration and expiration; and, therefore, ultimately upon the calibre of the bronchi in which the fluid is collected, and the force with which the respiratory act is performed. It may exist in expiration as well as inspiration, and may be diffused over a large extent of the chest, but its seat is usually the posterior and inferior portions of the lungs, in consequence of the frequency of catarrh and the predilection of that disease for that position. "There are many anatomical reasons which explain the frequency and persistence of the sub-crepitant râle at the base and posterior part of the chest. The bronchi are more numerous at the base than at the summit, and the chances of inflammation are therefore greater; they are longer, and the secreted fluids remain a longer time within them; their direction is different, and the disposition of the tubes is such that those of the upper parts free themselves sooner by expectoration, while those of the inferior portions empty themselves with much greater difficulty." Bronchitis usually terminates in the production of secretion, and therefore the sub-mucous, as the subcrepitant râle is sometimes called, is found associated with or consequent upon the sonorous and sibilous sounds. It must, however, be borne in mind, that the nature of the fluid poured out into the respiratory passages cannot be determined by the character of the subcrepitant râle. Writers speak of the mucous râle as if mucus produces a sound which is different in character to that resulting from the passage of air through pus, blood, or serum. The expression is, in my opinion, erroneous, and likely to lead an inexperienced auscultator to expect shades of difference which have no existence but in the imagination; the number of râles, bruits, and murmurs which really deserve mention, are sufficiently numerous without any necessity of increasing the catalogue by an excess of refinement which has really no practical value. The term mucous, or sub-mucous râle, is simply used, in consequence of the fact that 90 per cent. of the cases in which the subcrepitant sound exists, depend upon the presence of fluid secreted from the bronchial mucous membrane. Bronchitis may be general or local, acute or chronic,—when local, confined to the apex or apices, and persistent, we should be aware that subcrepitant râle usually indicates either crude tubercle, producing bronchial inflammation, or the same deposit passing into a condition of softening and gradual elimination. Lastly, the amount of subcrepitant râle audible in the chest is not always proportional to the quantity of the secretion contained in the air-channels, for the passages and vesicles may be so flooded with fluid that little or no air is capable of making its way through it. Such a result would be most likely to occur in acute or chronic œdema pulmonum, and would be recognised by the comparative absence of any vesicular or bronchial sound, by the dullness on percussion, and by a very moderate amount of subcrepitant râle. Dr. Sibson's chest-measurer would also show the comparative want of movement of the portion of the thoracic walls which corresponds to the œdematous lung.

Gurgling, or Cavernous Râle.—The character of this sound will depend upon the size of the cavity, the quantity of fluid contained in it, the freedom of the communications with the bronchial tubes, the force with which the air is made to traverse the fluid, and the nature of the walls bounding the space. Cavities which are formed by thin and elastic walls, contract easily during the act of expiration, and expel a portion, at least, of their contained air: they are, therefore, during inspiration, in a position to admit air, and to produce gurgling. On the other hand, cavities which are bounded by tough, tenacious, and non-elastic walls, can only expel their contents with difficulty, and are therefore incapable of producing a gurgling of such an intensity as in the former case.

From this consideration it follows, that the size of a cavity cannot be accurately known from the amount of gurgling heard in its interior; for a small active cavity (if I may so term it) may produce more noise and splashing than a much larger excavation, containing a quantity of air and fluid.

The cavernous râle, depending upon the same physical cause as the subcrepitant, cannot always be distinguished from it. The sounds pass and merge into each other, and, according to M. Barth, the characteristic difference between them is to be found in the presence of cavernous respiration. Now, as "rough or bronchial respiration often resembles cavernous respiration, especially when manifested at the root of the lungs," it is evident that M. Barth's mode of distinguishing subcrepitant from cavernous râle cannot always be depended upon; or, in other words, this author virtually admits the fact, that a clear distinctive line of demarcation cannot be drawn between the two sounds. Gurgling, therefore, is only to be regarded as a proof of the presence of air and fluid in a large space, whether bronchial or artificially formed, in the parenchyma of the lung. It occurs in bronchial dilatations, pneumonic vomicæ, and in the large hollows which succeed the elimination of softened tubercular matter. It is heard in the death-rattle, and whenever fluid accumulates in the larger portions of the respiratory tract. The preceding and concomitant symptoms, the persistency of its character and position, must aid us in detecting the pathological cause to which this abnormal sound is owing.

Consonating Râles.—When referring to bronchial respiration and bronchophony, I entered fully into the nature and theory of consonance, and showed how sounds which occur in one part of the respiratory tract may be conveyed to another portion of the same, and become strengthened and increased in intensity. The same principles will be found applicable to the various sounds which arise from the passage of air through fluid, with the exception of the vesicular crepitation. Whenever a portion of lung becomes consolidated, the bronchial tubes, which traverse a portion so diseased, will be in a condition to reinforce any sound propagated into them from another portion of the lungs; but as consolidation is the result of the vesicular texture being completely filled by morbid deposit, and such a condition renders the production of vesicular crepitation impossible, it necessarily follows, that simple crepitus can never be increased by consonance, and can be only heard at its seat of origin. All the modifications of the subcrepitant or gurgling sounds may, however, be propagated to, and distinctly heard in, a portion of consolidated lung, which is in free communication with the points from which the abnormal sounds proceed—a circumstance of considerable importance in diagnosis. I have, at this moment, a distinct recollection of the case of a medical student, who died from an acute attack of tubercular pneumonia, or, as Rokitansky termed it, infiltrated tubercle. The whole of the left lung presented a perfectly dull sound on percussion, with bronchial respiration and bronchophony of the most marked kind. Over the entire surface of the diseased side a distinct subcrepitant râle was audible, so loud and distinct in its characters as to give the idea of the whole lung being riddled by small cavities. On *post-mortem* examination, it was found that two or three cavities of small size existed at the apex, while the remainder of the lung was converted into a solid mass of grey hepatisation, no cavity, however small, being discoverable below the apex. The subcrepitant râle, therefore, in this case, produced in the upper portion of the lung, was propagated through the bronchial channel which traversed the consolidated part, and was thus rendered audible and distinct to the ear placed at any other point of the diseased side. Consonating râles have, therefore, the same signification as bronchial respiration and bronchophony: they prove that the air-cells of the part of the lung in which they are audible are filled with solid matter, and that a free and continuous channel exists between the source of the sound and the interior of the bronchial tubes, which traverse the consolidated pulmonary parenchyma. To distinguish between a consonating and non-consonating sound, we have only to call in the aid of percussion, and to bear in mind, that a dull sound, indicative of the infiltration of the lung with solid matter, or a tympanitic tone, characteristic of the presence of hollow cavities, are the conditions which attend the production of consonating vibrations.

There is one more form of crepitation which I must mention, termed by Laennec dry crepitating râle from large bubbles, its tone being compared by that author to the noise made by the sudden inflation of a dried pig's bladder. This râle has been abandoned by the majority of the French and English writers upon auscultation, but has found an advocate in Prof. Skoda, who considers it to be characteristic of vesicular emphysema.

The lung-cells, which in this disease have lost their elasticity, collapse without contraction during the act of expiration, and, on sudden inflation during inspiration, produce, by the rapid expansion of their walls, this peculiar sound. I am aware that subcrepitant râles occur in emphysema, but I am inclined to believe that they result from the presence of secretion in the dilated air-cells and terminal bronchi; and I cannot conceive the pulmonary membrane which forms the lung-cells to be so dry in emphysema, as to be capable of crackling upon expansion like a dried pig's bladder. Bronchial catarrh almost universally accompanies vesicular emphysema, and the secretion, which is consequent upon that affection of the lining membrane of the air-passages, will sufficiently explain the presence of the râle. In speaking of a modification of this sound, the character of which is fine, dry, and small, and which Skoda considers may occur in a slight dilatation of the air-vesicles, he remarks, that "he knows not how it is possible to distinguish this sound from that caused by tenacious mucus in the air-cells and smaller bronchial ramifications;" and I believe that the same observation may be applied with justice to the "râle crepitant sec à grosses bulles ou craquement," of Laennec.

Pleuritic Rubbing.—I have now to describe the last species of crepitus which is audible in the chest, and the nature of which can be explained in a few words. You are aware that the pleura costalis and pleura pulmonalis are moistened in the healthy state by a thin exhalation, which enables them to glide over each other without producing any appreciable sound. That there must be a movement of one pleural surface over another is evident from the mode in which the walls of the chest are expanded and the lungs inflated with air. The descent of the diaphragm and elevation of the thoracic parietes will evidently cause the pulmonary pleura to move in a downward direction over the pleura costalis during the act of inspiration, and in a reverse manner during expiration. As the general movement of the walls is greatest at the middle and lower parts of the chest, it will follow, as a necessary consequence, that the motion of the pleural surfaces over each other will be at a maximum in those positions; and in confirmation of this view, we find pleuritic rubbing very commonly developed in the situations just named, a result which is partly to be ascribed to the frequent deposit of false membrane in those regions of the chest.

The character of the sound is very easily recognised; sometimes soft, it resembles the noise made by the passage of pieces of dry silk over one another. MM. Barth and Roger very aptly compare it to the sound produced by rubbing with the finger the back of a hand placed over one's ear. Very frequently the sound is rough in its tone, as if some hard bodies were rubbing each other; the French writers then term it a *râclement.* It is usually heard during the act of inspiration; sometimes with the expiration; occasionally during both periods, when it is termed the to-and-fro rubbing sound, or the *frottement ascendant et descendant.* Its tone is rarely continuous, being rather a succession of crackles, like the sound made by walking on dry snow. The vibrations produced by the friction are often so distinct as to be capable of being felt by the hand placed over the diseased side. In all cases the sound appears to originate immediately beneath the ear, or the extremity of the stethoscope; its loudness depending upon the amount of solid matter exuded upon the pleural membranes, the force of the respiratory act, and the capability of the lung for expansion. Its duration rarely exceeds a few days; in some instances, however, and especially in those circumscribed pleurisies which attend tubercular deposits in the apices of the lungs, I have known the sound to be constantly present for months. The loudness and fulness of the respiratory murmur are usually diminished when the sound is present, but rarely changed in their quality. The diseases to which the rubbing sound is to be ascribed are:—pleuritis, tubercular deposit under the pleural membrane, and according to some writers, intervesicular emphysema. The mode in which the sound is produced in the first two affections I have already described. I have merely to add, that it can only occur when the membranes are not separated by any fluid, and that its presence is therefore an indication of the very commencement of pleuritis, or of the stage of recovery when the fluid has become absorbed, and the inflamed surfaces have again come into close contact. With respect to emphysema producing sound by the presence of blebs of air under the costal pleura, I cannot lay claim to any experience, and I am inclined to doubt whether such a condition would be capable of eliciting sound.

Cavernous Respiration.—No real distinction of kind exists between cavernous and bronchial respiration; the sounds differ from each simply in degree, and merge so gradually into each other, that in many cases considerable difficulty exists in deciding the real nature of the abnormal sound—its claim to be termed cavernous or bronchial. Laennec himself states that the two sounds have a similarity of character, and that a description cannot be given which can at all times and under all circumstances distinguish them. Position, the presence or absence of cavernous cough and râle, the co-existence of amphoric resonance, he states, will often assist to establish the distinction between them—a statement which evidently proves that the two sounds can present but slight differences in kind. I do not mean to say that there is no difference between a slight increase of respiratory sound due to a moderate degree of consolidation of the pulmonary substance and the murmur of a large and hollow cavity, but that an intense bronchial souffle frequently presents characters very similar to, and very difficult to distinguish from, cavernous respiration. The condition essential to the production of the abnormal murmur is the presence of a large hollow cavity surrounded by firm walls, and directly communicating with the larynx and bronchi, while the distinctness and fulness of the sound will depend upon the size of the cavity, the quantity of fluid contained in it, the number of bronchial tubes having a free communication with its interior, the nature of its walls in respect to their capability of reflecting sound, and the healthy or consolidated state of the pulmonary substance between the hollow space and the thoracic walls. The theory of the production of the sound is similar to that which I have already given for bronchial respiration. According to Dr. Sibson, "whenever an extensive cavity exists in the lungs the respiratory movements are restrained over that cavity, but not obliterated;" and again, "the respiratory movements over the region of dullness surrounding a cavity are much smaller than those over the cavity itself." Hence we may conclude that the air in a cavity is only partially changed at every act of respiration, and that the loud murmur heard over it is, for the most part, due to the air in its interior, consonating with the sounds produced in the upper part of the respiratory tract. Those cavities which are small in size, or whose walls are formed of comparatively healthy pulmonary tissue, or which communicate imperfectly with the bronchi, or which contain a considerable amount of fluid, offer conditions unfavorable for consonance, and therefore produce sounds which are weak and indistinct in their character. We see, then, that the size of a cavity cannot always be determined by the auscultatory signs which it presents; and you must be prepared to find that excavations of the pulmonary structure may sometimes be discovered in *post-mortem* examinations, whose real extent had not been clearly revealed during life by the cavernous murmur. The causes producing cavities in the lungs are various—tubercular softening with elimination of the morbid deposit, pneumonia terminating in abscess or gangrene, gradual obliteration and obsolescence of the proper vesicular texture of the lung, followed by bronchial dilatation, or the same result consequent upon chronic bronchitis, and an impaired elasticity of the bronchial mucous membrane. The diagnosis must be determined by the history of the case, the nature of the signs affected by the other means of examination, and by the position in the lung where it is most distinctly heard. In the large percentage of cases, (and we may say almost universally,) cavernous respiration at the apex of a lung will be found to be a sure indication of the presence of a cavity due to tubercular destruction of the pulmonary parenchyma.

LECTURE IX.

AMPHORIC RESONANCE. METALLIC TINKLING. STETHOSCOPE.

In my last lecture I stated that the sound which was termed by Laennec cavernous respiration could not be always considered pathognomonic of the presence of a cavity, in consequence of the occasional difficulty, and even impossibility, of drawing an accurate and well-defined line of demarcation between it and an intense bronchial souffle; and that the same remark could be equally applied to pectoriloquy and bronchophony. Amphoric resonance possesses, in my opinion, a far better claim to the title of cavernous, inasmuch as it is never found to occur but in those cases where large cavities exist, filled with air. The sound, as its name imports, resembles in its character the hollow murmur obtained by blowing or breathing forcibly into a large empty jug, or amphora, which has a narrow neck, and walls capable of reflecting any waves of air which may impinge upon them. The resonance has, in many instances, a humming kind of character, very similar to the noise made by the buzzing of a bee in a large vase, and was consequently styled by Laennec, the *bourdonnement amphorique.* A metallic tone is, also, often associated with this peculiar resonance, a condition found to be more frequently attendant upon the sound of the patient's voice. The amphoric resonance may exist of every degree of intensity, be permanent, or disappear at intervals, become gradually developed with the progress of disease, or make its appearance with extreme suddenness and rapidity. Its position is, in some cases, limited to the upper part of the thoracic cavity; but, in the greater number of instances, it is found diffused over the larger portion, or even over the entire surface of one side of the chest. During its presence all vesicular murmur is abolished over the space where the resonance is heard. With this exception it may be found associated with any of the sounds, normal or abnormal, produced within the cavity of the thorax. Hence we may meet with amphoric respiration, voice, and cough, the principles which explain one being applicable to the others. Whatever theory be adopted, all writers admit the existence of a large cavity, bounded by smooth and even walls, and capable of reflecting the vibrations of the contained air, to be a condition absolutely essential to the production of this peculiar reverberation. The size of the space requisite is not easy to determine, but I believe that the cavity must be at least equal to a moderate sized fist. The large excavations in the pulmonary substance, which succeed to the softening and elimination of tubercular matter, and the cavity formed between the pleura costalis and pulmonalis, in cases of pneumothorax, are the only conditions in which we are likely to meet with amphoric resonance in practice. In the former disease, a communication naturally exists by means of one or more bronchial tubes, between the outer air and the interior of the abnormal space, of sufficient extent to admit of the entrance of vibrations propagated along the air-channels from the other portions of the respiratory tract. With respect to pneumothorax, however, some difference of opinion exists, of the necessity of a fistulous communication between the lung and the cavity of the pleura, for the production of the resonance. This affection is, in the vast number of cases, the undoubted result of a perforated condition of the pleura covering the lung, but it is equally certain that the fistulous opening (if really present) is often so small as to escape the most diligent and scrutinising examination. The majority of writers upon the subject maintain the opinion, that an orifice is essential, asserting the phenomenon of amphoric resonance to be due to the reverberation, in the abnormal cavity, of the sounds produced in those bronchial tubes which communicate with the pleural space by means of the fistulous orifice, and they refer the occasional intermittence of the sound to the circumstance of the opening becoming at times blocked up by a quantity of fluid matter collected in its interior. On the other hand, Skoda and his followers deny the necessity of the orifice, resting their opinion upon the fact of the occurrence of pneumothorax, attended by amphoric resonance, in cases where no communication could be detected after death, and which, if really existing, must have been too small to admit of the passage of vibrations capable of producing such a sound as the amphoric resonance. They also refer to the following experiment:—If a stomach be inflated with air, and an individual speak or blow with some force through a stethoscope placed on any portion of its surface, the sound heard by another person examining the stomach at the same time will present a tone very amphoric in its character. In such a case the sound is evidently propagated to the contained air, across the elastic membrane of the stomach, and becomes increased by consonance, in the mode which I have already so frequently described. In the same manner, any sound produced in the lungs, being transmitted across a non-perforated pleural membrane, becomes increased in its intensity by the consonating vibrations of the air contained in the pleuræ, and in this way obtains its amphoric tone. MM. Barth and Roger admit the possibility of the truth of this view; for, in reference to an experiment exactly analogous to the one just mentioned, they add, "We shall be, consequently, less disinclined to admit the opinion of Dr. Skoda, relative to the production of the amphoric voice, a view which does not consider the communication of an abnormal cavity with the bronchial tubes to be an essential condition, but which attributes the phenomenon to consonance in such a way that the sonorous vibrations of the voice, directly transmitted through the pleura, determine consonating vibrations in the air effused between its laminæ."* A further corroboration of the theory here laid down is given in the following statement made by Dr. Addison, who certainly had not the above explanation in view at the time he wrote this observation:—"When pneumonic consolidation takes place anteriorly and inferiorly, and even posteriorly, on the right side, a remarkable degree of resonance upon percussion is occasionally elicited in a highly tympanitic condition of the intestines. Under precisely similar circumstances auscultation may detect a well-marked modification of amphoric respiration, and metallic tinkling to a considerable height in the chest; thereby leading to the erroneous conclusion that pneumothorax is present."* The occurrence of the phenomenon in such a case Dr. Addison explains, by supposing that the respiration owes its amphoric character to its conduction to, and reverberation in, the inflated stomach and bowels, and that the metallic tinkling is a sound originally produced below the diaphragm, and acquiring its intensity by reverberating in the opposite direction. The statement is, perhaps, difficult to understand, without a slight notice of the very interesting case upon which it is founded.

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* Barth and Roger, 'Traité Pratique d'Auscultation,' p. 213.

A girl, aged 19, admitted into Guy's Hospital, presented an increased resonance upon percussion, anteriorly, as high as the third rib, with dullness of the same lung, posteriorly. The respiration was puerile at the apices, and the sounds, consequent upon the acts of breathing, speaking, or coughing, on the right side, as high as the third rib, were attended with amphoric resonance—metallic tinkling was also present.

Post-mortem examination of the body showed that extensive adhesions of the pleuræ existed on both sides of the chest, and that the diaphragm, on the right side, had been raised up high within the thoracic cavity, partly by an inflated state of the bowels, and partly by the presence of old adhesions between it and the base of the lung. "A vast fæcal abscess extended from the pelvis to the under surface of the diaphragm on the right side. From this inflated abscess, or from the distended intestines, or both, had originated the great resonance, the amphoric sound, and tintement metallique."

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* Dr. Addison, 'Guy's Hospital Reports,' vol. iv, 1846.

The case is one of exceeding interest and importance, inasmuch as it proves the caution required to form a sound diagnosis of any affection of the contents of the chest. We can scarcely expect to have such puzzling cases frequently presented to us in practice. At the same time, it is as well to be on our guard, and to remember the possibility of such a group of symptoms as above given.

Reverting to the theory of amphoric resonance, I believe that in many, and perhaps the majority of, instances of pneumothorax, the morbid phenomenon is due to the reverberation in the pleural cavity of sounds which are directly conveyed to it through a fistulous opening in the pleura, but that it may, in many cases, also depend upon the transmission of vibrations from compressed or healthy lung, across the non-perforated membrane into the same cavity. Whatever theory be adopted, the practical point to be remembered is, that amphoric breathing, voice, or cough, indicates the existence of a tubercular excavation, or of the presence of air in the pleural space. Perforation of the membrane may undoubtedly arise from gangrene of the lung, from the bursting of a pneumonic abscess into the pleural cavity, from the corroding effects of a long-standing pleuritic exudation, and even from the rupture of cells which have become distended in an emphysematous condition of the lung; but such instances are rare in comparison with the perforation consequent upon tubercular disease; and we may therefore consider the sound to be more particularly associated with phthisis pulmonalis.

Amphoric resonance, which results from the presence of a tubercular cavity, is usually confined to the upper part of the chest, and gradually increases in definition and fulness, with the progressive enlargement of the excavation. Its tone is rarely fully developed, even in the largest cavities hollowed out of the pulmonary substance, partly in consequence of the roughness of the boundary walls, and partly from the presence of a quantity of secretion in the interior of the abnormal space. The resonance is generally associated with gurgling and subcrepitant rattle. Percussion usually elicits the bruit du pôt fêlè, and inspection exhibits a flattened condition of the chest wall, situated over the cavity, which presents a marked contrast with the rounded state of the parietes in pneumothorax. In the latter affection, the amphoric resonance is usually remarkably distinct—its seat is the general surface of one side of the chest, but in many cases it is confined to the mid-regions of the cavity of the thorax, in consequence of the presence of adhesions, which, uniting the pleuræ at the apex, prevent the passage of the air to the upper part of the space between the opposed folds of pleural membrane. The resonance is, in many instances, almost instantaneously produced and attended by an accession of sudden and violent dyspnœa, and as effusion of fluid is the ordinary sequence of perforation of the pleura, succussion of the patient will be generally attended by a splashing sound—true tintement metallique. Lastly, percussion yields a loud, sonorous, tympanitic sound over the portion of the pleural space containing air, while the lower part of the cavity emits a tone which is dull in a degree proportional to the quantity of fluid effused.

Tintement Metallique—Metallic Tinkling.—You are no doubt acquainted with the peculiar hollow, ringing, metallic character which the voice or any sound acquires in a large space filled with air, and surrounded by smooth, reflecting walls. Every one must be familiar with the tone obtained by striking and speaking into an empty cask, and the ringing sound of the foot-fall in a large passage or vault. The metallic resonance heard in the chest is evidently closely allied to the amphoric variety, and should be considered as a modification of it, being dependent upon similar conditions, and explained in the same manner. Ordinary metallic tinkling reverberation, or echo, is simply the most complete resonance capable of being obtained in the chest, and can only be elicited in cases where there exists a large cavity, filled with air, and provided with smooth, reflecting walls. There is, however, a variety of the sound, which deserves mention more by reason of the peculiarity of its character, than by any real importance which it might be supposed to possess. Laennec very aptly compared it to the noise made by the dropping of grains of sand into a hollow metallic vessel, and explained its production in the chest by supposing it to be occasioned by the reverberation in the pleural cavity of the sound resulting from the fall of drops of liquid into a quantity of effused fluid.

It is scarcely worth while to occupy your time with an account of the several theories which have been advanced to account for this metallic tinkling, and the resonance with which it is usually associated. Perhaps no abnormal sound within the chest has been the subject of greater discussion than the one we are considering, and certainly to an extent disproportioned to its importance. One writer, M. Raciborsky, in opposition to Laennec's view, which I have already given, ascribes the tinkling to the agitation of the fluid contained in the cavity of the pleura, under the belief, that some drops, becoming detached from the general mass of the effusion, fall back upon the liquid, and produce the peculiar resonance. However possible such an agitation might be, during a violent fit of coughing, it is not very clear how the result could take place during the simple act of respiration. Another writer believes the phenomenon to be due to the existence of a small fistulous opening below the level of the effused fluid, and that the air which enters the liquid from the lung bubbles breaks, and reverberates in the hollow space above the fluid. How the fluid level can be maintained at a height above the opening is certainly unexplained; nor, if the sound depends upon the entrance of air from the lungs, why the metallic tinkling should occur in expiration as well as inspiration. A third writer (Dr. Dance) supposes the occurrence of a still greater physical impossibility, by making two orifices, in place of one, in the pleural membrane. The streaming of the air through the orifice which is above the level of the fluid produces amphoric resonance, while the bubbling and breaking of the air entering the effusion from the opening below the fluid level accounts for the occurrence of the metallic tinkling. The laws of hydrostatics unfortunately invalidate this ingenious supposition. Skoda refers the sound to the consonance of the voice, breathing, and abnormal râles in the cavity of the pleura, at the same time admitting that drops falling from a height into a quantity of effused fluid may produce the phenomenon in question. I am inclined to admit Laennec's view entirely, and can only add, that while metallic resonance (erroneously called metallic tinkling) is a sound by no means infrequent, the true metallic tinkling—the sound of grains of sand falling into a hollow vessel—is of extremely rare occurrence. The conditions requisite for its production can only be found in pneumothorax and empyema, tubercular excavations, possessing rarely sufficient size and smoothness, to lead to the development of this remarkable sound.

Succussion or Fluctuation Sound.—It is a remarkable fact in the history of medicine, that while this sound was known and described by Hippocrates more than 2000 years ago, the profession should have remained in ignorance of the existence of sounds in the interior of the chest, and of the means of detecting them, until the nineteenth century. The idea of listening directly to the chest of a patient appears to have occurred to the mind of the great father of medicine; for in attempting to distinguish between hydrothorax and purulent effusion, he says—"Thus you will know that the chest contains water and not pus—by applying the ear for a considerable time to the sides (*καὶ ἢν πολλον χρονον προσεχων το οὖς ακουαζη προς τα πλευρα,*) you hear a sound similar to the vibration of boiling vinegar,"*—an observation, which, however valueless with respect to the diagnosis attempted, proves that Hippocrates was aware of the production of sound in the cavity of the thorax. According to the interesting historic notice of MM. Barth and Roger, we learn that Cælius Aurelianus, and in later times, Harvey, appear to have had some glimmerings of auscultation; and a passage is quoted in which the discoverer of the circulation of the blood speaks of the movements of the heart being attended by sound, especially at the moment when the blood passes from the venous into the arterial system. The notice further gives the sarcastic and ignorant rejoinder of Æmilius Parisanus, a Venetian physician, and author of an attempted refutation of the views of our illustrious countryman, who, denying the existence of the phenomenon, speaks of it as a sound to be heard only in London—*tantummodo Londini exauditur.* Reverting to the subject from which we have digressed for a moment, we find the following passage in the works of Hippocrates respecting succussion:—"After having placed the patient upon a solid and immovable seat, let his hands be held by an assistant, and shake him by the shoulders, in order that we may hear on which side sound will be produced." And again:—"Of patients attacked by empyema, those in whom much sound is heard have less pus than those in whom the sound is less and the respiration more constrained." The statement is perfectly correct, although the *rationale* of the fact appears to have been unknown to Hippocrates himself. He was not aware that air and fluid must coexist in the cavity of the pleura to produce this fluctuation sound. A splashing noise cannot be obtained from a full cask when shaken, nor will the cavity of the pleura emit a fluctuation sound unless air and fluid coexist. Succussion is, therefore, a means of detecting pneumothorax with empyema, and the gurgling noise elicited by its performance is so characteristic that it may be in some cases recognised by the patient himself, and heard at a considerable distance from his chest. It is very frequently associated with true metallic tinkling, and almost invariably with amphoric or metallic resonance. Its duration is variable—cases are on record where it has been distinguished for years. The sound has been stated by some authors to have been found in large pulmonary excavations partially filled with fluid. Such a result is possible, but I have never met with it. In my opinion the fluctuation noise is characteristic of pneumothorax with empyema.

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* Hippocrates. De Morbis, ii. Vanderlinden.

Auscultation of the Cough.—A few words may be advantageously added respecting the diagnostic signs afforded by the act of coughing, although it must be admitted that their value is far inferior to those presented by the voice and respiration. The principal advantage of the act consists in the increased force and rapidity which it impresses upon the air traversing the bronchial channels and lung cells, and its consequent effect in rendering the sounds, already described in the course of these Lectures, more audible to the auscultator. A quantity of secretion, lodged in the bronchial tubes, may arrest for a time the formation of sounds in the portion of the lung below the obstruction, or prevent the transmission of sounds (respiratory or vocal) produced in the upper part of the respiratory tract. A forcible act of coughing, by removing the obstruction, enables us to detect the real condition of the lung. Again, many patients under examination have no idea of the mode of breathing; and from the manner in which they perform the respiratory act, an inexperienced student might consider the greater portion of their lungs to be impermeable to air, while in fact the pulmonary organs are in a perfectly healthy condition. The long inspiration which precedes and succeeds the act of coughing, inevitably introduces the air into the smallest ramifications, and informs us of the permeability or impermeability of the air channels. In the same way crepitation, which is almost inaudible in ordinary breathing, becomes marked and distinct by making the patient cough; and the same remark applies more especially to the gurgling sound peculiar to excavations, which contain a quantity of air and fluid. Independent of what has been already stated, the sound produced by the act of coughing presents some varieties worthy of mention. In the healthy state of the chest the sound is dull, confused, spread over the general surface of the thoracic parictes, and accompanied by a certain vibration of these walls. The noise is most marked where the bronchi are the largest in size, when the sides of the chest are thin and elastic, and the force of the act of coughing the greatest; the alterations impressed upon its character by disease are exactly analogous to the changes effected in the respiratory sounds and voice. The cough may be bronchial, tubular, cavernous, amphoric, or metallo-resonant. And as I have entered at some length into a description of the causes which similarly modify the sounds produced in breathing and speaking, I shall not trouble you with any further remarks upon the subject, except to state that the amphoric variety may be easily imitated by coughing into a large jug, when a humming metallic resonance is obtained, exactly similar to this kind of cough.

I believe that I have now given an account of the sounds, normal and abnormal, which may be found to occur in the pulmonary organs. I have traced out, as far as possible, their causes and practical signification; and, although I am aware that I have, in many instances, repeated the same observation, I believe that I have only done so when its importance demanded its full impression upon your minds. I shall now conclude this division of my course by describing a method of examining the chest, which is more curious than important,—I mean, a branch of auscultation termed, by a French physician, *autophony*, or *heautophonics*,—and then make a few general remarks upon the Stethoscope, a subject which, I find, I omitted to mention at the commencement of this course. The science and value of autophonics may be dismissed in a few words. By the term is meant the art of examining a patient's chest by an observation of the manner in which the voice of the auscultator is affected by the condition of the contents of the thoracic cavity. I fear that this explanation requires an elucidation of its own. An illustration will, however, explain its meaning best. Place the palm of the hand over your ear, and when speaking you will immediately observe that the vibrations of the voice appear to be damped, and to impress a degree of unpleasant resonance, very different to the natural sensation experienced in speaking. A thin sheet of paper placed over the ear produces an effect different to that of a solid body of greater thickness. Reasoning on this fact, M. Hourmann conceived that the autophonic resonance would be augmented in cases of increased density of the contents of the thoracic cavity;* and it must be admitted, that in many cases a consolidated condition of lung will produce such an effect; but the difference is too slightly marked, and the number of exceptions too great, to allow us to attach any importance to the science. I have never been able to detect any difference between the autophonic reverberations accompanying pneumonic consolidation and pleuritic effusion, and am therefore entirely in accord with MM. Barth and Roger, who disregard the new mode of exploring thoracic diseases. They add, that "they are not the only individuals who have derived scarcely any result from the new process. MM. Bouillaud, Priory, and Raciborsky, have experimented, and have come to the same conclusion, that the voice of the auscultator experiences, in the morbid state, no other modification but a resonance, which is perhaps a little more marked on the diseased side, but destitute of any special character."

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* Revue Médicale, 1839. Barth et Roger, p. 180.

Another method of examination has been still later proposed, a combination of auscultation with percussion, the chest being struck at the same time that the effects of the blow upon the contained viscera are attempted to be distinguished by the application of the ear to some other portion of the thoracic wall. I have frequently tried to learn something of the condition of the pulmonary organs by this mode, but have never been able to obtain any satisfactory result.

Theory of the Stethoscope.—Although the causes which produce sound in the chest are evidently very different in their nature—viz., the friction of air against the internal surface of the bronchial and vesicular channels, the bursting of bubbles in those passages, the rapid flow of blood over smooth or roughened membranes, or through orifices of normal or abnormal size, the sudden tension of valves, and the impulse of solid bodies upon each other; the vibrations which are recognised by the employment of auscultation, mediate or immediate, are simply those which are communicated from the thoracic parietes. The internal organs impress their vibrations upon the general surface of the chest, and constitute it the medium by which their physical conditions are to be distinguished. Whatever may be the character of the sounds in the chest, *per se,* we can only know them by the vibrations which they establish in the walls of that cavity; and our aim is to determine the most efficient means of making them sensible to our nerves of hearing. Now, by a fundamental law in acoustics, we know that a sound is best heard in the medium in which it is produced. Sounds originating in air are most distinctly heard in air, those produced in water heard best in water, and the same for solids. I have not time to illustrate this principle, although many interesting examples might be adduced in proof of its truth. It will be sufficient, for my purpose, to remind you, that as sound is most readily conveyed when the number of media through which it has to travel are as few as possible; and when the media resemble the original sounding body as closely as possible in elasticity and density, the vibrations of the thoracic parietes will be communicated to the ear in the fullest and clearest manner by the direct application of that organ to the surface of the chest. Immediate auscultation ensures the most perfect conduction of the sounds; but as many reasons exist which render this mode of examination in many cases disagreeable, inconvenient, and sometimes improper, we are compelled to employ some instrument which may answer the same purpose. Stethoscopes have been constructed of all kinds of material, and of endless varieties of form, and may be solid or hollow, rigid or flexible. Wood, metal, compressed gold-beater's skin, bone, gutta-percha, wire surrounded by Indian rubber, and numerous other substances, have been employed as materials, but none have been found capable of superseding the light specimens of cedar wood. The varieties of form have been without number, some due to principle, a few to ideas of elegance of shape, and many more to convenience, and portability. I am in the habit of employing the common hollow stethoscope, and do not attach much importance to its shape. I should recommend an instrument which, while it is not hurtful to the patient by the pressure required in its use, is at the same time most comfortable to the ear of the auscultator. My stethoscope is made of one piece of wood, and has one end considerably smaller than the other, for the purpose of examining the jugular vein, and the hollow space above the clavicle which corresponds to the apex of the lung.

In discussing the principles of the hollow stethoscope, we have to determine whether the sounds which are communicated by its means to the ear are conveyed chiefly through the central column of air, or through the solid parts of the instrument—a subject which has ever been a source of contention among writers on auscultation. The majority of writers, following the opinion of Dr. C. J. Williams, (to whose labours in this field the profession is so deeply indebted,) ascribe the active power of the instrument to the column of air contained in its interior, and which they suppose to be set into vibration by the portion of the chest wall covered by the hollow of the stethoscope.

One writer, (Mr Barrett,)* pursuing the idea to its fullest extent, considers the subject mathematically, and has even calculated the form of a stethoscope which should concentrate and transmit the greatest amount of these chest vibrations to the ear. He suggests that the expanded portion of the instrument should be constructed in the form of a paraboloid, in such a manner as to have its focus in the centre of commencement of the shaft, and that the shaft should be hollowed out into the shape of an ellipsoid, having its foci respectively coincident with the focus of the paraboloid, and the centre of the upper orifice of the instrument. By such a contrivance, the vibrations which fell upon the concave end would be concentrated in the common focus of the two figures, and emanating from that point, impinge upon the ellipsoidal surface, to meet again at the other focus, over which the ear of the auscultator would be placed. The idea is very ingenious, and looks well on paper, but would be found useless in practice, as no instrument could be easily turned to this shape. Independently of this mechanical difficulty, the instrument would require the rays of sound to be parallel to the axis of the paraboloid, in order that they might meet at the focus—a condition which would render the instrument comparatively useless. We shall, moreover, find that the column of air contained in the stethoscope is not set into vibration in the manner described.

The annexed diagram will serve to explain a few simple experiments which clearly illustrate this subject, and of the truth of which you can readily convince yourselves with the instrument before you.

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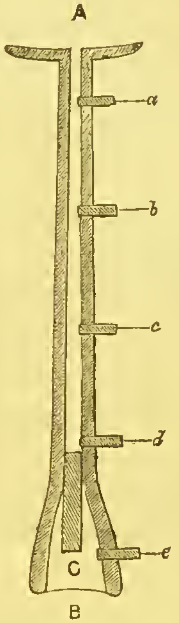
* 'Medical Gazette,' New Series, vol. i., 1837-8.



A B is a vertical section of a Stethoscope.
a b c d e, small orifices in its shaft.

Experiment 1. The holes being closed by small wooden pegs, the instrument, becoming a common hollow stethoscope, conveyed, when placed over a healthy chest, a full and distinct respiratory murmur to the ear.

Exp. 2. The holes being opened one by one, the murmur became gradually and, when all the orifices were open, remarkably diminished in its distinctness and fulness; a weak murmur did not reach the ear.

Exp. 3. The insertion of a plug into the shaft at C, only slightly impaired the distinctness of the murmur.

Exp. 4. The plug remaining in the instrument, a remarkable diminution in the intensity of the sound followed upon the removal of the small pegs.

Exp. 5. When a plug was introduced at A, or a solid earpiece employed, the respiratory sound became distinctly audible. The opening of the orifices, and the closure of B by a plug, in this case produced little or no difference in the character or intensity of the sound.

Exp. 6. A solid stethoscope was found to communicate sound very readily to the ear, although not quite so perfectly as the ordinary form.

Exp. 7. The insertion of a plug into the chest end of a flexible stethoscope, impaired, but did not destroy, the conducting power of the instrument.

From the consideration of these experiments we gather the following conclusions regarding the hollow stethoscope :—

1. That its conducting power principally depends upon the column of air confined in its interior, for the opening of the holes in the shaft, by allowing the divergence of vibrations, prevented the communication of the vibratory sounds to the ear.

2. That the central column of air is not chiefly set into vibration by the portion of the chest wall included under the hollowed end of the instrument, for a thick plug introduced into the lower end of the shaft only slightly impaired the conducting power of the stethoscope.

3. That the enclosed column of air can only owe its vibrations to those which are communicated to it from

the solid parts surrounding it, and that the sounds of the chest are, therefore, in the first instance, propagated through the wood to the air within the tube, and thence, by reason of the continuity of the columns of air, into the interior of the external ear.

I do not mean to assert that the solid walls have no share in the direct communication of the sound; we have seen that a solid stethoscope conducts sound very readily, and that the opening of all the orifices does not entirely destroy the power of the instrument. The experiments which I have made will, however, serve to show that the conditions of the hollow and solid stethoscopes are not analogous, and that the two forms must be explained on different grounds.

Lastly, it is evident that the flexible stethoscope conducts the sounds of the chest to the ear through the contained column of air being set into vibration by the elastic walls which surround it.

LECTURE X.

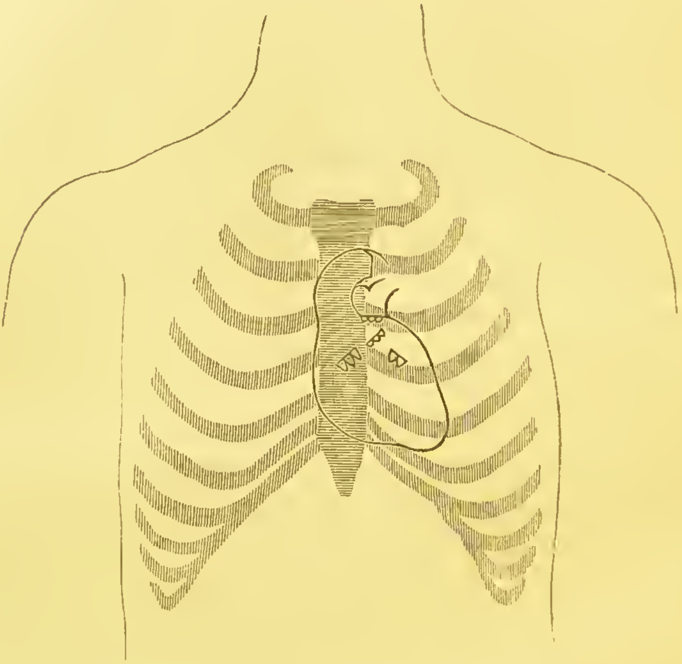
THE HEART.—POSITION.—MEASUREMENTS.—INNERVATION.—IMPULSE.

No very accurate data can be given respecting the absolute and relative frequency of the Diseases of the Heart and its great Vessels, for the number of deaths reported by the Registrar General, as the result of these causes, affords a very imperfect idea of the real importance of this class of cases.

In the tables published for 1846, while the mortality from all causes is estimated at 49,277, the number of deaths ascribed to cardiac affections amounts to 1783, a proportion which is evidently too small, when we remember that a number of the cases classed under the terms Dropsy, Disease of the Lungs, Cerebral Apoplexy, and other affections resulting from a disturbed balance of the arterial and venous systems, are the simple physical consequences of a diseased condition of the muscular substance of the heart, or of the orifices through which the torrent of the circulation is unceasingly passing. It would be useless to attempt to form any accurate estimate of the absolute or relative frequency of the diseases to which the heart and its investing sac are liable. I have merely alluded to the subject in proof of the importance of the study of this portion of our course, and of the necessity of an intimate acquaintance with this branch of physical diagnosis.

As the general anatomical description of the heart and its investing membrane, the pericardium, are too well known to require any description here, I shall simply content myself with referring to such points as appear to be more peculiarly connected with our subject, viz., the determination of the position of the outline of the heart and the situation of its orifices, the nature of the nervous power which regulates its movements, and the mode in which its healthy impulse is produced. The boundaries of the space within which the normal heart performs its movements must be evidently subject to some variation, partly in consequence of the motion of the organ in systole and diastole; partly from the alterations in the capacity of the chest, due to the acts of inspiration and expiration; and partly from the effect which the different positions of the body impress upon the situation of the viscus. The limits of this space cannot be expected to be capable of being laid down with rigorous exactness; but for all practical purposes, we may consider the outline traced in the accompanying diagram to be a sufficient approximation to the truth.

The heart is seen in the sketch to lie behind the middle and lower bones of the sternum, extending slightly on the right side, beyond the sternal extremities of the third, fourth, and fifth costal cartilages, and situated on the left side of the chest, behind the cartilages of the third, fourth, fifth, and sixth ribs. Its left border is received into a deep hollow formed in the left lung; its base lies on a level with the junction of the cartilages of the third ribs with the sternum, and its apex strikes the thoracic wall, at a spot between the fifth and sixth ribs, situated nearly two inches below the nipple, and about one inch to the left of the sternum.



Posteriorly, the heart is separated from the bodies of the sixth, seventh, and eighth ribs, by the œsophagus and descending aorta, while anteriorly it is overlapped by the right and left lungs in such a manner, as to leave a small lozenge-shaped portion of the pericardium uncovered by pulmonary tissue. The upper and lower boundaries of this space are, the fourth and sixth costal cartilages respectively, and its inner side corresponds to the mesial line of the sternum. Its area varies with the degree to which the lungs are inflated, and at the moment of extreme expiration rarely exceeds 2½—3 square inches, while its longest and shortest diameters may be calculated at 3 and 2 inches respectively.

Our diagram will also serve to exhibit the actual and relative positions of the orifices of the heart—a point of considerable importance to the auscultator. With a little latitude, we may fix their positions as follows:—

1. The tricuspid orifice is situated immediately behind the sternum, on a level with the articulations of the cartilages of the fourth ribs with that bone.

2. The mitral is on the same line of level with, but in a plane posterior to, the tricuspid opening. It lies on the left of the sternum, and immediately behind the cartilage of the fourth rib.

3. The orifice of the pulmonary artery lies on a level with the junction of the cartilages of the third ribs with the sternum, so that a needle passed through the left edge of that bone will pierce its valvular apparatus. The orifice is nearly three quarters of an inch higher than that of the tricuspid.

4. The aortic opening is situated behind and somewhat below the orifice of the pulmonary artery,—slightly beyond the left edge of the sternum, and in the space between the cartilages of the third and fourth ribs.

5. The pulmonary artery in its ascent from the right ventricle, passes behind and somewhat to the left of the sternum, and at the distance of nearly two inches from its origin, divides into its right and left branches.

A needle passed close to the sternum in the space between the cartilages of the second and third ribs, will, after traversing a layer of lung of nearly an inch in thickness, penetrate the vessel.

6. The aorta springs from the base of the left ventricle, and ascends immediately after its origin in a direction from left to right, as far as the right edge of the sternum, close to the junction of the cartilages of the second and third ribs with that bone, being covered in this part of its course by the right lung and pericardium. From this point the artery curves to the left side and posteriorly, and passing behind the centre of the first bone of the sternum, lies upon the trachea a little above the origin of the bronchi, and subsequently upon the second and third dorsal vertebræ. A needle passed perpendicularly through the first bone of the sternum, will penetrate the arch of the aorta, the upper border of which, in the adult, is about 10—12 lines distant from the notch at the top of the sternum.

A knowledge of the measurements of the heart is of considerable importance to the student, and according to M. Bizot, the best authority on the subject, the—

	Men.	*Women.*	
Length of the Heart is equal to	$43\frac{3}{23}$	$41\frac{2}{27}$	Lines.
Breadth ,,	$47\frac{18}{23}$	$44\frac{1}{27}$	,,
Thickness of the right Ventricle—			
,, base	$1\frac{39}{46}$	$1\frac{19}{27}$	,,
,, middle	$1\frac{7}{23}$	$1\frac{13}{54}$	,,
,, apex	$1\frac{45}{46}$	$1\frac{25}{27}$	,,
Thickness of the left Ventricle—			
,, base	$4\frac{17}{46}$	$4\frac{3}{27}$	,,
,, middle	$5\frac{1}{11}$	$4\frac{27}{54}$	,,
,, apex	$3\frac{13}{23}$	$3\frac{6}{27}$	,,

Circumference of—	*Men.*	*Women.*
Tricuspid orifice	$54\frac{5}{23}$	$47\frac{4}{27}$ lines,
Mitral	$48\frac{9}{23}$	$40\frac{17}{27}$,,
Pulmonary artery	$31\frac{12}{23}$	$29\frac{1}{3}$,,
Aortic	$30\frac{20}{23}$	$28\frac{1}{9}$,,

The weight of the heart varies with the age and stature of the individual. The following table gives the result of a number of examinations made on the subject by the late Dr. Clendinning:—

From 15—30 years of age,	the weight averages	$8\frac{1}{4}$ oz.
30—50 ,,	,,	$8\frac{1}{2}$,,
50—70 ,,	,,	$9\frac{1}{3}$,,
70 upwards ,,	,,	$9\frac{3}{4}$,,

Whence it is evident, that the heart is continually increasing in weight, although the other parts and organs of the body are becoming atrophied.

The effect of stature upon weight has not been subjected to such careful examination; but as a general rule, broad-chested individuals, although of small height, are found to have larger and weightier hearts than persons of greater height and less capacity of chest. For the description of the plexuses of nerves which supply the heart, I must refer you to your anatomical works. I will only quote the statement made by Dr. Kirkes, that "a number of minute ganglia of the sympathetic system, with connecting nervous filaments, are scattered through the muscular structure of the organ, and that while the ganglia act as so many centres or organs for the production of nervous power, the connecting nerve fibres unite them into one system, enable them to act in concert, and to direct their impulses, so as to excite, in regular series, the successive contractions of the several muscles of the heart."

This view is the more modern idea of the nervous arrangements of the heart, and is entirely at variance with the theory laid down by Haller and his disciples. According to the opinion of this eminent physiologist, every muscle possesses an inherent power called contractility, which only requires the presence of its proper stimulus to make itself evident by the contraction of the fibres. The heart, like any other muscle, is endowed with this power, the blood being the peculiar means of exciting this irritability, and the contractions of the organ exhibiting the result of the stimulus. Several objections present themselves to this theory :—(1), that a heart deprived of its blood will continue to pulsate for some time after the removal of its supposed stimulus; and (2), that dilatation is found to take place in a heart maintained filled with blood, and in which the stimulus to contraction is therefore constantly present. To the latter objection Fontana has replied, that the relaxed condition of the ventricles is the simple consequence of the exhaustion of the muscular irritability due to previous contraction, and he accounts for the rhythm of systole and diastole upon the supposition of an alternate exhaustion and reproduction of muscular contractility or irritability. Volkmann, however, very properly urges, that when the parts of a living body are continuously subjected to a stimulus, one of two consequences must follow,—either the stimulus is so great that exhaustion and death follow very rapidly upon its application, or else the organ reacts no longer against its influence with the same amount of energy as previously. The constant stimulus of the will, or of an electric rotation machine produces a contraction which terminates with exhaustion, when any further action of the stimulus becomes impossible. An alternate play of contraction and dilatation cannot occur under such circumstances, and hence the experiment which is related by Haller himself, of a heart contracting and dilating subsequent to the tying of the aorta, and when the blood remained in its interior, serves to a great extent to invalidate his own theory. In fact, the effect of mental impressions upon the action of the heart, the arrest of the pulse, and the dilatation of the ventricles when the par vagum is brought within the circle of an electro-magnetic apparatus, the retardation of the ventricular contractions in persons under the influence of digitalis, and lastly the elaborate provision of nerves with which the organ is supplied, are sufficient proofs that ordinary nervous power, and not muscular irritability, is essentially necessary to maintain that unceasing motion which the heart exhibits during the whole period of life.

We may proceed even further, and bring to our aid the facts supplied by the observation of disease, which prove how any irritation of the branches of the par vagum, distributed over the stomach and pulmonary organs, become propagated by the cardiac branches of that nerve to the heart, and their effect shown in an irregular action of that organ. Fully admitting, then, as a fundamental doctrine, the view that the movements of the heart depend upon nervous power, we find the opponents of Haller at issue among themselves respecting the share which the medulla oblongata possesses in the production of that influence; and Volkmann proceeds still further to show, that the heart derives its contractile energy from the presence of the ganglia scattered through its muscular substance. "The medulla oblongata," says this experimentalist, "is one of many conditions of the heart's movements, but not the most important and proximate. Wilson, Philip, and Flourens showed that circulation was possible after the destruction of the spinal cord. Bidder proved that the blood in frogs continued to move eleven weeks after the same operation. Dr. Reid instances the case of an animal living thirteen days after the section of its nervi vagi, and Morgagni one of eighteen days." Volkmann saw the circulation in a frog seven days after the central nervous organs had been completely destroyed. Such experiments, says this author, prove that the heart can remain a long time irritable without any connection with the medulla oblongata, and that any nervous influence necessary to produce muscular contraction must be principally derived from the ganglia situated in the substance of the organ. These ganglia are independent sources of nervous power, each, in all probability, regulating a certain portion of the heart, and all being so connected by nervous filaments, as to work harmoniously together in producing that synchroneity of action which characterises the movements of the organ. Disturbing causes may destroy, to a certain extent, that unity of action, and give rise to those remarkable irregularities in the number of sounds, of which we shall have to speak hereafter, just as in a frog which has been recently killed, the heart may be seen performing its movements in a most irregular manner,—the auricles acting disproportionately fast to the ventricles, and the latter contracting alternately instead of synchronously with each other.

Leaving this vexata quæstio of physiologists, let me direct your attention to another knotty point, which has been equally the subject of much controversy. I mean the nature and mechanism of the heart's impulse against the chest wall. The chief point at issue among authorities upon the theory of the stroke, is, whether the heart has a movement of projection, a species of swinging forwards in systole, to which the impulse is to be ascribed, and if so, to what force this motion is due, or whether the impulse is the simple result of the spirally twisting movement of the heart upon its long axis. Mr. Cruveilhier founds his opinion upon a remarkable case of malformation, observed in a child, born at the Maison Royale de Santé, at Paris, in July, 1841.* He states that the heart was exposed without a pericardium, and presented a dry and smooth surface, that touching it neither disturbed its action nor produced any symptoms of pain in the child, that its position changed with every movement of the body, and that it became vertical when the child was placed in the erect posture.

The following are a few of the observations detailed:

1. That there exist only two periods in the movements of the heart,—the one of contraction, the other of dilatation, and no period of repose for the entire organ. The ventricles having ceased to act, the auricles immediately commence their functions.
2. That the diastole of the ventricles is effected in one half the time required for the performance

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* 'Gazette Médicale,' August, 1841.

of their systole. "The ventricular contraction is not accompanied by a movement of projection of the heart forwards, it is the contraction of the heart in the spiral manner, which alone causes the apex of the organ to approximate to the thoracic walls."

3. That during the ventricular systole, the summit of the left ventricle describes a spiral or corkscrew motion from right to left, and from behind forwards, causing the impulse.
4. That the ventricular dilatation is accompanied by a movement of the heart downwards, which in the case related was so marked that the impulse was thought at first sight to occur during the period of the diastole.

These statements being the results of direct observation, would appear to be incontrovertable. Let us notice, however, another view, which is equally the result of observation on the living body. Professor Skoda* says:—"In May, 1847, I examined a child of a few days old in whom the sternum was absent, and whose chest presented a fissure narrow above and wide below, and simply covered by skin. With every inspiration the skin was drawn deeply towards the vertebral column, while the anterior ends of the ribs were bent somewhat inwards. With every expiration, the skin was driven forward in the form of a bladder. By palpation, the heart could be felt lying in a vertical position, moving in a direction downwards and forwards in systole, and upwards and backwards in diastole. The stroke was felt at every systole directly above the insertion of the diaphragm, and at every diastole at the height of the second rib, when the finger was passed sufficiently deep towards the spine. The stroke of the diastole was just as strong as that of the systole. By placing two fingers at such a distance that the lower one felt the systolic and the upper the diastolic impulse, it was found that the heart glided through a space of nearly one inch, in a downward direction. The outline of the heart was seen when the skin was moderately distended, and it could be discerned, that the impulse felt at the points mentioned resulted, not from an enlargement or lengthening, but from a shifting or displacement of the heart. When the skin was not touched, the outline of the heart during systole was observed during an expiration in an elevation, which passed from above downwards along the puffed out skin; while in diastole a depression could be traced along the same surface, traversing it from below upwards. During inspiration the circumference of the heart could be seen in systole and diastole whenever the skin was pressed towards the vertebral column. The heart moved tolerable accurately from above downwards in the middle of the fissure, when the child was placed upon its back. When the child was turned towards one side, the heart moved from the median line, towards the side upon which the body lay."

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* Skoda; Abhandlung über Perkuss. und Auscult., p. 149, 4th Edit.

It is sufficiently evident that the statements of these two great authorities are diametrically opposed to each other, and I see no means of reconciling their conflicting observations. Skoda's case was one better adapted for examination, as the child was older, and its condition less abnormal, than that described by Cruveilhier, whilst in the latter the eye could more easily trace the course and order of the heart's movements. Let us, however, study the manner in which Skoda explains the supposed projection of the heart forwards and downwards in systole, and see how far other facts will substantiate his view. The theory advanced by him is so ingenious and simple in character, as to merit considerable attention, and I think deserves to be brought before your notice, as no English writer has yet fully described the view which is now adopted and warmly advocated by the present distinguished medical school of Vienna. The able work from which the above theory is taken has already reached its fourth edition, and should be in the hands of all those who are sufficiently familiar with the German language to be able to master its contents, and who are anxious to become thoroughly acquainted with the physical diagnosis of the diseases of the chest.

The heart, according to this theory, is supposed to have a swinging motion in systole, which causes it to move in a direction downwards and forwards, at the same time that the muscular contraction producing this movement alters the shape of the organ, and tilts up its apex. The swing by which sufficient momentum is acquired to produce the characteristic impulse, is simply the movement due to the recoil of the heart, the principle of which may be briefly explained in the following manner :—Suppose a vessel of any shape to contain a quantity of fluid (liquid or gaseous), which exerts a pressure upon every portion of its internal superficies. Then, so long as the vessel is closed on all sides, the pressure being equal in all directions will give it no tendency to move in any particular direction.

But suppose a part of one of its sides to be suddenly removed, and a free passage allowed for the exit of the contained fluid,—the equality of pressure upon two opposite sides of the vessel will be destroyed from the loss of pressure at the orifice, and the greater force excited upon the internal surface of the vessel opposite to the opening, will tend to move the vessel in a direction the reverse of that of the issuing fluid. A vessel with a narrow base containing fluid may be upset by allowing its contents to escape from an opening in one of its sides. The greater horizontal pressure opposite to the opening tends to tilt the vessel, and if the base be narrow will entirely overthrow it. The well known Barker's mill acts upon the principle of recoil, or the inequality of pressure upon two opposite surfaces. The motion of an ordinary rocket is, however, the most familiar example of this kind of pressure. The rocket is a tube containing explosive materials, which generate, when kindled, a quantity of gas within its interior. By the evolution of the gas, a considerable amount of pressure is produced, and for a time maintained, upon every part of the internal surface of the rocket; but as an escape of the gas is constantly taking place through an orifice at one end of the tube, the pressure upon the inner surface of the end in which the opening is situated, will be less than the pressure upon the inner surface of the opposite extremity of the missile. Hence, the greater pressure upon the upper part of the interior of the rocket will cause its ascent in a direction opposite to the course of the issuing gas, until the weight of the tube is greater than the force of the pressure, when the rocket will evidently commence to fall. The analogy between the motion of the heart and the rocket will now become apparent. The blood in the ventricles being powerfully compressed in the moment of systole, reacts with a corresponding force upon every portion of the inner surface of these chambers; and on the valves being suddenly thrown back by the rush of the blood through the orifices of the aorta and pulmonary artery, the pressure at these outlets is reduced to nil, and the non-counterbalanced pressure upon the surfaces exactly opposite to the orifices tends to swing the entire organ in a direction downwards, forwards, and to the left side. As the two ventricles, if free, would move in directions exactly opposite to the currents which pass through the orifices of the aorta and pulmonary artery respectively, the motion of the entire organ will be in a direction the resultant of the two just mentioned; but as the reaction pressure in the left may be considered to be three times greater than the similar force in the right ventricle, it will follow that the movement of the apex of the heart will be principally in a direction opposite to the flow of the stream through the aortic orifice. Such is the view, somewhat in extenso, advocated by Professor Skoda.

To establish a probability of its correctness, we should possess some definite idea of the force actually exerted by the systolic contraction of the ventricles, so that the amount of the reaction pressure or recoil force may be estimated. The calculation of this power has engaged the attention of many distinguished physiologists, who have arrived at results ridiculously at variance with each other. Thus Borelli estimated it at 180,000 lbs., Bernouilli at 3000 lbs., Keil at 5 oz., Jurin at 9 lbs., our ingenious countryman, Dr. Hales, at $51\frac{1}{2}$ lbs., and an eminent French physiologist at

4 lbs. 3 drachms. It would be out of place in these Lectures, to attempt to trace the causes which have led to such an extraordinary contrariety of opinion; but it may afford some little interest, and be at the same time instructive, to observe the steps by which an estimate of the force may be fairly obtained. The best account of the process is contained in a remarkable treatise upon Hæmodynamics, recently published by Dr. Volkmann, one of the Professors of the University of Halle.* As the very basis of our calculation, we must remember that every fluid, in passing through a tube, exerts a two-fold power,—one in the direction of the long axis of the tube, producing the current, and measured by the rapidity of the stream; the other acting in a direction perpendicular to the sides of the tube, and striving to expand and burst them. This principle is of universal application, and is, therefore, true in every part of the vascular system of the body. Hence it is evident, that the contractile power of the heart has a two-fold object in every artery.

(1.) To overcome the resistances offered by the parietes of the vessel to the passage of the blood,—a force which may be measured in any vessel by introducing a tube into its side and observing the height and weight of the column of blood impelled into it by the action of the heart.

(2.) To produce and maintain the velocity of the blood streaming through its channel.

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* Die Hæmodynamik nach Versuchen von Dr. Volkmann. Leipzig, 1850.

To measure, therefore, the heart's force in the stream which traverses the aorta, we must calculate the amount of the resistance overcome by the current of the blood, and also the force which is requisite to produce the velocity of the fluid in that vessel—for which latter purpose we must be previously informed of the velocity with which the current of the circulation pours through the aorta. The sum of the two forces will represent the power exerted by the contraction of the left ventricle.

(1.) Now, by Poisseuille's Hæmodynamometer, it was inferred from analogous experiments, made upon living animals, that the blood which would pass into the instrument when introduced into the carotid artery of a man, would sustain a column of mercury of 160 millimetres in height. Hence, if from a comparison of the capacities of the aorta and carotid artery, we suppose the pressure in the latter to be ¾ths of the similar force in the former vessel, it will follow that the powers required to overcome resistance in the aorta will be represented by a column of mercury nearly 200 millimetres in height.

(2.) To determine the force required to produce the velocity of the blood in the aorta, which, on the authority of the best physiologists, we may estimate at 470 millimetres per second, we have only to substitute the numerical value of the velocity in the equation, $f=\frac{v^{2}}{4g}$[*]; where f is the unknown force, we are calculating $v=$the volocity, and $g=$ the force of gravity $=32$ feet $=9830$ millimetres.

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[*] If an orifice be made in a vessel containing fluid, the level of which is preserved at a constant height, the velocity of the issuing fluid will

Hence,
$$f = \frac{(470)^2}{4 \times 9830} = \frac{220900}{4 \times 9830} \text{ millimetres}$$
$$= 5{\cdot}7 \text{ millimetres};$$

i. e., if we suppose an opening to be made in a vessel containing mercury, at a depth of 5·7 millimetres from the surface of the fluid, the stream which issues from the orifice would possess a velocity of 470 millimetres per second, or the velocity of the blood in its passage through the aorta. Hence a column of mercury 5·7 millimetres in height, represents the force exerted by the left ventricle in producing velocity.

Combining, therefore, the two forces,—of resistance and velocity,—we find that the entire column of mercury which the force of the left ventricle is capable of sustaining in systole, is one which has the area of a section of the aorta for its base, and its height=205·7 millimetres.

Hence this power = weight of (area of aorta × 205·7) cub. in. of mercury,
= ,, 908 × 205·7 ,,
= ,, 11·2 cub. inches of mercury,
= ,, 5·4 lbs. avoirdupois, nearly.

This is called the statical power of the left side of the heart; and using Breschet's language, is the force with which the blood moves in the artery,—that is to say, if a diaphragm were placed across the artery and maintained on one side by a pressure of 5·4 lbs.,

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evidently depend upon the depth of the orifice below the surface of the level.

If v = velocity of the issuing fluid,
d = depth of orifice from surface,
g = force of gravity.

$v^2 = 4gd \; \therefore d =$ depth of orifice required to produce velocity (v), $\frac{v^2}{4g}$.

Hence the velocity being known in the aorta, the height of the column of blood required to produce it will be easily calculated.

against the current issuing from the left ventricle, the blood would cease to move in the artery.

I have taken the area of a section of the commencement of the aorta at 908 square millimetres,—hence, reduced to English measurement, the pressure during systole, upon every square inch of the internal surface of the left ventricle, equals nearly 4 lbs.,—and if the right be considered to contract with one third of the power exhibited by the left ventricle, the pressure, *i. e.*, the power of recoil exerted upon the parts of the two ventricles, which are exactly opposite to the outlets of the aorta and pulmonary artery, will equal a statical pressure of nearly $5\frac{1}{3}$ lbs. avoirdupois. Such being the force which acts almost instantaneously, (for the systole is usually commenced and completed in $\frac{1}{140}$th part of a minute, the pulse being supposed to beat 70 times per minute,) is it unreasonable to imagine that a force of this amount could impress a movement of recoil upon an organ which, on the average, only weighs ten ounces, and hangs loosely suspended in the pericardium, from a number of highly elastic yielding vessels? I confess the view appears very feasible, and I must further add, that upon any other theory than that of recoil, I cannot understand how the heart becomes capable of producing those violent impulses against the chest which it occasionally exhibits.

The spiral twisting from right to left movement of the heart round its axis, however strongly exerted, cannot produce a direct blow upon the chest. No screwing, however forcible, could produce those violent shocks which we know the heart frequently imparts to the chest wall.

This theory of Skoda's which I have attempted to carry out into calculation, has met with decided opposition from the eminent physiologist Valentin, who states, that having snipped off the apex of a frog's heart, he observed the motion of the organ persisting for some period; that the heart of an animal will frequently move after the blood has been removed from its interior; and that no analogy can be drawn between the self-creating expansive power of the gas in a rocket, and the reacting pressure of the blood upon the inner surfaces of the ventricles during systole.* These objections, however, appear to me to be of slight value, for no one doubts that the muscular fibres of the heart will contract upon the application of stimulus, even subsequently to an injury by a section across them; but we have no proof of a true systolic impulsive stroke being made by the little heart of the frog, after the mutilation it had received; and I cannot understand, on any theory whatever, that any twisting or tilting motion could be expected after the continuity of the spirally-arranged muscular fibres had been destroyed. With respect to the movement of the empty heart after its removal from the body, no one has described it as capable of producing a distinct impulse. To the last objection I can only answer that, so long as a strong reaction pressure really exists, the effects will be the same, in whatever way the pressure is produced. The different modes in which the recoil pressures are obtained in Barker's mill, rockets, and fireworks, does not affect the identity of the theory which explains their action by a principle common to them all. That this pressure exists, is proved by the simple experiment already referred to, as made by Dr. Hales, who found that blood rose to the height of eight or ten feet in a glass tube, inserted into the carotid artery of a horse; and it is curious to observe how the result of the calculation I have made, exactly tallies with the statement made by this clever experimentalist. He estimated the power of the left ventricle at 51½ lbs. From the results at which we have arrived, the pressure is calculated at 4 lbs. upon every square inch of the inner surface of that ventricle, and as the internal superficies of that chamber averages about thirteen square inches, our result of 4 × 13 = 52 lbs. accords very remarkably with the estimate of the ingenious writer.

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* Valentin. Lehrbuch der Physiologie der Menschen.

In the account which I have thus given of this part of the physiology of the heart, I have no doubt travelled somewhat out of the proper province of these Lectures; but as diagnosis is based upon pathology, and the latter upon an accurate knowledge of the mode in which the functions of organs are discharged, I think that an excuse may be easily found for bringing this theory before your notice. The subject is undoubtedly very intricate in nature, and I fear to some of my hearers, who are not familiar with mathematical language, I may have only added a little more mystery to what was before dark and obscure; but I think the theory should be better known in this country than it is at present, not alone in consequence of its ingenuity, but also as it is now the prevailing doctrine of the distinguished medical school of Vienna.

I must not dismiss this subject without stating, in a few words, the more common view adopted in this country, and which is to be ascribed to Dr. Hope.

The theory is as follows:—

(1.) That the auricles—more especially the left—forming the posterior part of the base of the heart, the aorta and pulmonary artery constituting the anterior,

(2.) Become the fixed points to which the muscular fibres of the ventricles contract;

(3.) And that the contraction during systole towards the origin of the great vessels, draws the tense and rounded body of the ventricles upon the auricles as a fulcrum, by which means the apex is tilted upwards against the chest-wall, and the stroke produced.

A fundamental objection to this theory resides, in the fact, that the auricles cannot act as a fulcrum, in consequence of being themselves moveable and dependent, from elastic vessels which open into their cavities. And supposing such a fulcrum to be possible, the auricles are not sufficiently filled during the time of the ventricular systole, to offer that solid, unyielding resistance, which is the very requisite of a fulcrum. In a word, the walls of their cavities are too thin, their chambers are too little distended with blood, and the whole of that portion of the heart too moveable, to admit a belief in the lever explanation of the stroke, which is advocated in the late Dr. Hope's admirable work.

LECTURE XI.

INSPECTION.

Having discussed at some length certain physiological points respecting the Heart and its great Vessels, I shall now commence the consideration of the practical part of the subject, and adopting the plan laid down in the first division of this course, proceed to describe the physical signs of cardiac diseases, presented by—

A. *Inspection.*
B. *Palpation.*
C. *Percussion.*
D. *Auscultation.*

In the course of the description of the several signs, I shall take the opportunity of briefly sketching the various physical lesions to which they are related.

A. *Inspection.*—There can be no doubt that the outline of the heart may be detected, in many normally constructed chests, by a prominence which is situated to the left of the sternum, and that the lower boundary of the organ may be traced (as described by Dr. Sibson) in a depression, which crosses the sixth and seventh cartilages of the left side. In the majority of cases, however, the most careful inspection fails to distinguish "any accurate stamping of the bulk and outline of the heart upon the thoracic wall;" and with the exception of the gentle elevation and depression of the part corresponding to the apex, the cardiac region presents no remarkable bulging or prominence of its surface, sufficient to indicate the presence of the organ from which its name is derived.

The position of the heart's impulse varies in health within certain limits. Thus, in children and women it beats comparatively higher than in men: in the former, in consequence of the preponderance in size of the abdominal over the thoracic organs, and in the latter, in all probability from the pressure of the stays contracting the diameters of the lower portion of the chest. In tall, thin individuals, with long chests and wide intercostal spaces, in persons advanced in age, whose hearts, as we have already seen, increase in weight, even when the remaining organs of the body become atrophied, the apex, or rather its impulse, is found to assume a somewhat lower position in the chest than ordinary. The erect posture also favours this lowered position of the impulse. In fat persons on the contrary, the most attentive inspection will often fail to detect the point at which the heart comes into contact with the chest wall; while the same result is sometimes observed in thin persons, whose hearts are healthy in tissue, but possess a very small amount of contractile energy.

The principal abnormal circumstances which may be observed by the Inspection of the surface of the chest, corresponding to the heart and large vessels, are:—

1. A double stroke in the præcordial region.
2. A double stroke, præcordial and sternal.
3. A displacement of the apex of the heart.
4. An unusual prominence of the præcordial region.
5. A movement of undulation.

1. The peculiar phenomena of the back stroke or diastolic impulse is one of considerable interest, and has engaged the attention of many distinguished writers upon the diseases of the heart. In cases where it occurs, the normal elevation of the fifth intercostal space, during the ventricular systole, is seen to be followed by a similar elevation of the fourth intercostal space during diastole. Two distinct impulses are observed, respectively synchronous with the ventricular contraction and dilatation, at two distinct points of the præcordial region, and thus the upper and lower parts of the anterior surface of the heart appear to exhibit a species of alternate or see-saw motion. According to Dr. Hope, the superior or second impulse is never observed in the normal state, nor in simple dilatation of the heart. It may occur in all cases of simple hypertrophy, but more commonly in eccentric hypertrophy of the organ, and its cause, according to this author, is "the diastole of the ventricles, during which the heart suddenly sinks back from the wall of the chest with a force greater in proportion to its thickness and capacity." Admitting most fully the truth of the statement, that the second impulse can only occur in cases of hypertrophy and dilatation, I confess that Dr. Hope's theory gives me no definite idea of the mode in which this abnormal action of the heart is produced. May we not rather

suppose that the enlarged heart, laterally displacing the right and left lungs, becomes more closely applied to the chest wall, and that the sudden expansion of its right ventricle during the moment of diastole, produces the peculiar impulse? The cases quoted in my last Lecture, tend to exhibit the force of the diastolic expansion; for in the one given by Cruveilhier, the diastole was so marked, that the impulse was at first sight thought to take place during this period of the heart's action; and in the instance described by Professor Skoda, the diastole appeared to be as strong as the systolic impulse. Whatever be the nature of the action by which the diastole is effected, all writers agree in the fact of the suddenness and force with which the expansion of the previously-contracted chambers is performed. Thus if we consider the diastolic period to equal one half of the systolic, and the latter to occupy one half of the time from pulse to pulse, it follows that the ventricular expansion requires only $\frac{1}{4} \cdot \frac{1}{75}$th of a minute $= \frac{60}{300} = \frac{1}{5}$th of a second (supposing the pulse to beat 75 times per minute) for its completion,—a smallness of result leaving no doubt of the rapidity, almost instantaneous, with which the ventricles are refilled with blood. That an action so rapid, by which the ventricular walls are suddenly thrown outwards, should be performed with considerable force, is not only probable, *per se*, but also confirmed by the cases to which I have already referred; and if further corroboration were necessary, the experiment of firmly grasping, with both hands, the heart of an animal in vivisection, would show that the compressing power, exerted by the experimenter, must be excessively great to prevent the diastolic expansion. Hence, if such be the result in a normal condition of the heart, may we not expect a considerable increase of the expansive force, when the organ has become the subject of hypertrophy and dilatation? and is it not reasonable to anticipate a marked impulse against the ribs during its diastole, when, by reason of its increased volume, the whole surface of the right ventricle is maintained during the systole and diastole, in a position of close proximity to the chest wall ? If this view be correct, it is evident that the see-saw, double stroke of the heart, becomes a valuable indication of eccentric hypertrophy, especially in persons whose chests being somewhat narrow, in the antero-posterior diameter, do not allow of much space for the abnormal enlargement of the organ. The intercostal spaces, in such individuals, are usually very wide, and the whole of the anterior surface of the chest considerably flattened.

2. In the normal condition of the chest, the aorta and pulmonary artery lie at a depth of nearly one inch below the surface of the sternum, being covered, during the moment of inspiration, by the overlapping portions of the right and left lungs. The natural pulsations of these vessels, which are thus imbedded in pulmonary substance, cannot, therefore, be expected to be conducted to the surface of the chest; and any dilatation which either may suffer, must be of considerable extent, to be capable of displacing the lungs, and by coming into contact with the parietes, of producing an impulsive stroke upon the intercostal spaces, right or left of the sternum.

From the extreme rarity of aneurismal dilatations of the pulmonary artery, we may pass at once to those of the aorta. The several forms which these abnormal enlargements of the vessel may assume, are, according to Rokitansky:—

(1.) Cylindrical.
(2.) Fusiform.
(3.) Unilateral.
(4.) Pedunculated.
(5.) Cirsoid, *i. e.*, a string of pouches placed alternately upon the aorta.—(Aneurisma Cirsoideum—Varix Arterialis.)

But whatever be its form, the morbid enlargement invariably invades the convex more frequently than the concave side of the aorta (ascending and transverse), and its direction of growth is always towards the right side of the chest, so that an extensive aneurism of the ascending portion may, in time, occupy the greater part of that side of the thoracic cavity.

The mechanical effects of the tumour are shown in the displacement and compression of the right lung, and by the gradual advancement to the anterior surface of the chest, where its presence becomes indicated by a strong impulse, synchronous, or nearly so, with the stroke of the heart. We have here, therefore, as in the last case, two evidently distinct impulses, at points which respectively correspond to the positions of the apex of the heart and of the aneurismal swelling; but in the one case, the impulses occur alternately, in the other synchronously, with each other. It should be borne in mind, however, that a considerable aneurism of the aorta, may exist without any pulsation being externally visible, and that the absence of this symptom does not negative the possibility of the presence of this morbid condition of the vessel.

Aneurisms of the descending thoracic aorta are developed more frequently upon the posterior and lateral, than upon the anterior portion of the vessel, and, in making their way towards the vertebral column, they produce caries and destruction of its substance. By a further extension of the disease, the aneurism spreads over the posterior wall of the left side of the chest, destroying the ribs, and, in some cases, even a portion of the scapula, so that upon inspection, a tumour may be found in the back, pulsating synchronously with the ventricular contraction of the heart. The skin over the swelling is often reddened, and generally acutely painful to the touch. An aneurism of the abdominal aorta, is often observed to be attended (especially in thin people) by a distinct elevation of the abdominal walls; but it should be known that this diseased condition of the vessel may exist without giving any signs of its existence by inspection, and that, on the contrary, very evident pulsations may be seen in the abdominal walls, when the artery is in a perfectly healthy state. The latter pulsations are either nervous in their origin, occurring in hysterical and easily excited individuals, appearing in paroxysms, and unattended by any abdominal swelling, capable of being detected by palpation and percussion, or they depend upon the presence of tumours, which involve and partly compress the aorta, and conduct its pulsations to the surface of the abdomen. A collection of flatus in the intestine will, occasionally, give rise to the production of these pulsations. The diagnostic marks of the several morbid conditions, which are thus attended by the same abdominal pulsation, will be determined by palpation and percussion, and by a careful examination of the history of the case. Considerable attention is often requisite in these cases to unravel their nature. A collection of hardened fæcal matter in the intestine, producing pulsation of the abdominal aorta, has been occasionally mistaken for actual disease of that vessel.

3. Much information may be obtained respecting the condition of the heart, from an Inspection of the position of its apex. The displacements of this portion of the heart may be to the right or left, above or below,—its normal position; or the entire organ may be so driven towards the vertebral column, by the pressure of fluid or solid matter, that no portion of its surface will be able to come in contact with the thoracic parietes.

(*a*). *Laterally.*—This displacement results, in the majority of cases, from the morbid accumulation of air or fluid, single or combined in the cavity of one of the pleuræ—(empyema, hydrothorax, pneumothorax).—Emphysema of the left lung tends to render the long axis of the heart more vertical, and at the same time diminishes the area of the exposed portion of the anterior surface of the organ, by the interposition of an unusual quantity of dilated pulmonary tissue between the heart and the thoracic wall. In such cases, the apex is frequently found pulsating in the epigastrium, especially in persons in whom the sternum is unusually short. Lateral displacement occurs also, when a portion of the lung, in which the heart lies imbedded, has become permanently diminished in volume, either from an atrophy and obliteration of the pulmonary tissue, consequent upon chronic pleuritic effusion, or from the contraction of substance resulting from chronic pneumonia. The structural changes in the lung being permanent, the altered position of the heart will be equally persistent.

(*b*). *Upwards.*—The heart, and consequently its apex, will be forced in an upward direction by all abdominal enlargements which impede the descent of the diaphragm. Pregnancy, ascites, tympanites, enlargements of the spleen and liver (particularly of the left lobe of the latter), evidently tend to elevate the position of the heart. Dr. Walshe states that the same result may ensue from the contraction and diminution of volume, attendant upon tubercular disease of the upper part of the left lung. I cannot say that I have been able to verify this statement, but I have no doubt of the possibility of the displacement from such a cause.

(*c*). *Downwards.*—The presence of a large quantity of exudation in the cavity of the pericardium, (Bouillaud has described cases, in which serous fluid has accumulated to the amount of seven pounds,) will tend most materially to lower the heart's apex,—

(*α*.) From the increased weight of the pericardial sac, depressing the portion of the diaphragm upon which it rests; and—

(*β*.) From the heart sinking, by reason of its greater specific gravity, to the lowest part of the exudation. Unless adhesions already exist between the opposite sides of the upper part of the pericardium, the fluid must accumulate towards the base of the heart, around the origins of the aorta and pulmonary artery, and by its continued soaking action impair the elasticity of the coat of these vessels, and thus still further increase the heart's tendency to maintain its depressed position. As a point of some interest, it should be remembered, that in extensive pericardial effusions, the heart often swings so loosely in the surrounding fluid, that the point where the apex strikes the præcordial region cannot be determined. Although abnormally depressed, the summit of the left ventricle appears to have a wider range for its impulse than it possesses in the normal state. In chronic cases, however, where the elasticity of the vessels has been considerably impaired, and partial absorption of the fluid has taken place, the body of the heart sinks in such a manner, as to render its long axis more than usually horizontal, and causing the apex to beat to the left of its normal position.

γ. Other causes of depression may be found in hypertrophy and dilatation of the heart, and in aneurismal diseases of the aorta; in emphysema pulmonum, sufficiently developed to be capable of pushing the diaphragm downwards; and in the sudden removal of fluid, which had been accumulated in the abdominal cavity.

(*d*). *Backwards.*—Emphysema of the anterior portion of the left lung, aneürism of the aorta, developing itself in front of the heart, cancerous, tubercular, and other tumours formed in the anterior mediastinum, repel the heart towards the vertebral column.

4. Supposing that there is no original malconformation of the chest, an unusual prominence of the præcordial region is indicative of either—

(*a*). The presence of a considerable quantity of fluid in the sac of the pericardium; or—

(*b*). An hypertrophy and dilatation of the heart.

(*a*). The rupture of the muscular tissue of the heart, or of an aneurism of the commencement of the ascending aorta, may lead to the sudden accumulation of blood in the pericardium. Death, however, follows too rapidly upon the effusion, to allow of any outward symptom of the fluid to appear in the præcordial region, even in cases where several pounds of blood have passed through the abnormal opening.

A small quantity of serum is always found in the pericardium after death, undoubtedly resulting from the condensation of the habitus, which separates the opposed surfaces of the sac during life. An abnormal accumulation of fluid of the same character—clear, yellowish, free from admixture with fibrinous flakes, and therefore not dependent upon an inflammatory condition of the investing membrane of the heart—is termed hydrops pericardii. The quantity effused may vary in quantity from a few ounces to several pounds; and the continued pressure which it exerts in the latter case may produce a prominent condition of the præcordial region. Serous effusion into the pericardium is usually associated with and dependent upon the same causes which lead to a general anasarca of the several cavities of the body.

The most frequent and important cause of an accumulation of fluid in the pericardium is, an inflammatory condition of that sac,—a disease, which, although commencing with the usual appearances of redness and injection, gives rise to exudations not only dissimilar in character, but productive of very different effects upon the general condition of the body. "These exudations consist either of substances which are already present in the blood itself, but in so excessive a quantity, that the normal condition of that fluid can be only restored by their elimination (primary exudations), or the morbid new products are the result of further changes in the original constituents of the blood (secondary exudations)."*

To the former class belong—

1. Plastic exudations.
    - (*a*) fibrinous.
    - (*b*) albuminous.
2. Serous.
3. Hæmorrhagic.

To the second class—

1. Purulent exudation.
2. Decomposed ,,
3. Tubercular ,,
4. Cancerous ,,
5. Secondary hæmorrhagic.

Whatever be the nature of the exudation, whether primary or secondary, its mechanical effect will entirely depend upon the quantity of fluid exuded, and the period of time during which it has been accumulating in the pericardial sac. The extent of the prominence being regulated, in some measure, by the elasticity of the præcordial portion of the thoracic wall, being, of course, more marked in children and young individuals, whose structures are more capable of yielding to pressure than in adults and aged persons. "The distension by fluid of the pericardial sac, besides displacing the surrounding organs, pushes forward the sternum and costal walls, elevates the second costal cartilage, and to a less degree the third, fourth, and fifth, widens the space between the cartilages and ribs, from the second cartilage to the seventh rib, projects outwards the sixth rib, and causes some degree of bulge over the left side, and some protrusion of the slope formed by the lower edges of the costal cartilages." (Dr. Sibson, 'Med. Gaz.,' vol. xli.) In many cases, the inflammatory action is propagated from the pericardium to the serous membrane which lines the intercostal muscles. A species of paralysis of these muscles is the direct consequence of this condition, by which their capability of resisting pressure becomes diminished, and the further extension of the morbid projection of the præcordial region favoured. This loss of mobility of the præcordial space becomes, therefore, a valuable index, in many cases, of the existence of pericardial inflammation, and affords a diagnostic mark between effusion consequent upon this lesion, and eccentric hypertrophy of the heart. We must, however, remark, that a large exudation may exist without this abnormal elevation of the præcordial region, and that, on the other hand, a prominence may be distinctly visible, and result from other causes, such as a large aneurism of the aorta developing itself in front of the heart, an effusion of fluid into the left pleural cavity, and lastly, from—

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* Zehetmayer.—Die Herzkrankheiten, p. 97.

(*b*). Hypertrophy and dilatation of the heart.

In this case, the presence of a large abnormal mass forcibly impinging upon the chest wall, is evidently stamped in outline upon the præcordial space. The strong and well-marked accompanying impulse at once distinguishing it from the thoracic elevation due to the presence of fluid in the cavity of the pericardium.

5. Pericardial effusion is occasionally marked by the passage of undulations from below upwards, across the præcordial region—the commencement of the wave being synchronous with the systole of the heart. "It has always been either between the cartilages of the second and third ribs, or of the third and fourth, or between both at the same time, that I have seen this motion, and never in any other situation."* The appearance of this phenomena is sufficiently indicative of the condition of the pericardium; but the sign is too frequently absent to be worthy of much consideration. In the case of a boy, aged thirteen years, I observed a similar undulatory movement, traversing the larger portion of the left side of the chest, and, from its extent and concomitant symptoms, clearly dependent upon the propagation of the heart's impulse, through fluid contained in the left pleural cavity.

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* Dr. Latham, on 'Diseases of the Heart,' p. 133.

LECTURE XII.

PALPATION.

By Palpation, or laying the hand upon the præcordial region, we are enabled to detect the position of the apex of the heart, and the force with which it comes into contact with the thoracic wall. Certain abnormal conditions of the endo- and pericardium may be also detected by this means of investigation. Of the normal and abnormal position of the impulse I have already spoken, and I have simply to confine myself, at present, to the consideration of the other signs of cardiac disease obtained by Palpation.

In a former Lecture I discussed, at some length, the theories offered by different writers in explanation of the mechanism of the stroke of the heart, and I stated, as the result of the several views, that the impulse may be considered to be due to the recoil of the heart and the tilting upwards of its apex in systole.

To these causes I might have added the lengthening of the aorta and pulmonary artery consequent upon the sudden projection of a quantity of blood from the ventricles into these vessels. If we consider the action of recoil to be the principal cause productive of the impulse of the heart against the chest-wall, it is clear that a full effect can be only obtained when a certain relation exists between the weight of the heart, the diameters of the arterial orifices, and the capacity of the ventricles. For the sake of convenience I shall call that portion of the internal surface of the ventricles, which is exactly opposite and equal to the orifices of those chambers, "the area of pressure," inasmuch as the force exerted upon it during systole chiefly determines the force of the impulse of the heart against the thoracic wall. There are, therefore, three principal circumstances to be considered in estimating this recoil effect:—

1. The weight of the heart to be moved.
2. The extent of the area of pressure.
3. The amount of the pressure of the blood in systole upon the ventricular walls.

An increased weight of the heart (I mean of its true muscular tissue) may be considered to represent a proportional increase of contractile force, and a consequent increased recoil effect upon the area of pressure. We have no means of measuring this increase of power; but, from the observation of the immense mass of muscular substance developed in an hypertrophied heart, we may fairly conclude that the extra development of contractile force will at least counterbalance the extra weight of the heart to be moved. We may, therefore, in the majority of cases, disregard the weight of the increased mass which is to be set in motion, and direct our observations more particularly to the extent of the area of pressure, remembering, however, that the pressure upon this surface during systole has been abnormally increased by the augmented development of the walls of the ventricles. And, in the *first* place, if the aortic outlet be narrowed by disease, the normal quantity of blood cannot be discharged through it in systole, unless the ventricular contraction be abnormally prolonged in duration, or the muscular power increased to augment the velocity of the issuing current. In the former case the recoil power will be below its normal amount, and the force of the impulse proportionally weak. In the latter the impulse will be, in most instances, stronger than normal, although not to any considerable amount, in consequence of the diminished area of pressure. I say, in most instances, for exceptions exist in which the impulse, in aortic contraction, is abnormally weak; in such cases the orifice being reduced by disease to a mere chink, the area of pressure is equally reduced in size, and the recoil pressure becomes, in spite of the increased contractile power of the heart, unable to produce a forcible movement of impulse. The organ rolls about in the chest, but fails to impinge upon the walls of that cavity with its former characteristic stroke. In a moderate amount of stenosis or contraction of the aortic orifice, the existence of the concomitant hypertrophy of the walls and dilatation of the left ventricle usually increases the force of the impulse, at the same time that the period of the ventricular contraction is ordinarily observed to be prolonged.

Secondly. If the aortic outlet be normal in size and the left ventricle increased in volume and augmented

consequences of hypertrophy or atrophy, and that each of these states is divisible into three varieties:—

1. Simple	Hypertrophy or Atrophy.
2. Concentric	
3. Eccentric	

That the chambers may be abnormally enlarged in capacity from—

1. Eccentric hypertrophy; or—
2. Eccentric atrophy, with a normal, diminished, or enlarged volume of the heart.

And that the chambers may be abnormally diminished in capacity from—

1. Concentric hypertrophy.
2. Concentric atrophy, with a normal, abnormal increase, or abnormal diminution in the thickness of the heart wall.

With respect to the last variety, it may appear contradictory to designate, by the name of atrophy, a condition in which the walls are of normal or even of abnormally increased thickness; but the anomaly vanishes when we remember that, in these cases, the entire volume of the heart and its weight, as shown in the balance, are considerably below the natural standards. The result specified proceeds from the shrinking and contraction of the heart in all directions, occasionally observed in some cases of atrophy.

Resuming our remarks upon the impulse, we find its force increased—

1. In the normal heart, as the consequence of mental emotions and of reflex irritations propagated through the pneumogastric and sympathetic nerves; of the frequent use of stimuli, or of the presence of inflammatory action in some organ of the body, especially the lungs; and, lastly, of any cause, such as bodily exertion, which propels an unusually large quantity of blood towards the right side of the organ.

2. In simple and eccentric hypertrophy of one or both ventricles to an extent proportional, *cæteris paribus*, to the morbid enlargement. The præcordial region, in such cases, is seen to elevate the hand or stethoscope, or head of the observer placed upon it, in a most distinct manner, and when the heart is considerably enlarged, the sound of the impulse to the auscultator acquires frequently a species of ringing character. Post-mortem examination shows that a distinct præcordial elevation in systole indicates either an eccentric hypertrophy of the entire heart, or an hypertrophy and dilatation of the left ventricle dependent upon an insufficient condition of the aortic valves. I have already stated why an excessive impulse should not be expected in contraction of the orifice of the aorta, although the left ventricle be considerably hypertrophied. From the smallness of the quantity of blood expelled at every systole, the force of the impulse is always inconsiderable in concentric hypertrophy. The weight of the heart, in this case, appears to be too great to be moved by the force excited upon the area of pressure.

Professor Skoda makes the following important observations upon the occurrence of an occasionally marked augmented impulse:—"The left ventricle cannot be expected to be always able to drive a large quantity of blood into the aorta, unless an equal quantity be received from the right ventricle by the pulmonary artery. Now, if the hypertrophy and dilatation be confined to the left ventricle, while the right ventricle preserves its normal size, or is even diminished in volume; the left ventricle will be incapable of propelling a considerable quantity of blood into the aorta, because an equal quantity is not received, in the same time, by the right ventricle; the contractions of which are synchronous with the left. Should there be no insufficiency of the aortic valves (in which case a portion of the blood expelled in systole would regurgitate into the ventricle in diastole) no augmented impulse could be constantly maintained in an eccentric hypertrophy limited to the left side of the heart, but only powerful strokes would be observed to occur from time to time. The same result is found in hypertrophy and dilatation of the right ventricle, with diminished or normal capacity and atrophy of the walls of the left ventricle, unless an insufficient condition of the tricuspid valve is present; and the force of the heart-stroke diminishes the more, the greater the disproportion between the capacities of the two chambers."

Lastly, it must be observed, that all causes which induce general debility, such as loss of blood, profuse discharges from mucous membranes, the imperfect supply of proper nutrition, &c., may so reduce the vital energy of the body as to render the impulse of an hypertrophied heart even weaker than the impulse of the organ in its healthy state. The interposition of an emphysematous portion of lung, between the enlarged heart and the chest-wall, will evidently break the force of the impulse, and mask, to a great extent, the actual condition of the organ. Hence, we conclude, that hypertrophy may exist to some amount without the sensation of a remarkable impulse or palpation, and that an augmented impulse does not necessarily indicate an enlarged condition of the heart. But an impulse, so marked as to be capable of raising the hand or heart placed over the præcordial region, may be taken to be a sure indication of a heart considerably enlarged in all its diameters.

3. In pericardial adhesions the impulse is frequently considerably augmented in force; the adhesions being cellular or fibro-cellular bands situated at the apex of the heart, along its sulcus transversalis, or around the origins of the arterial trunks. Those which more especially attach the apex to the opposite side of the pericardium must evidently tend to impede and shackle the free action of the heart, which increases its efforts to maintain its propelling power, and gives rise to an augmented impulse. By the unceasing action of the organ the bands are, in some cases, loosened and entirely separated from one of their points of attachment, and the heart freed from the restraint to which it had been subjected. Post-mortem examinations exhibit, in these cases, a mass of shreds of false membrane or cellular tissue left on one or both of the opposite sides of the pericardium. Where pericarditis has terminated in a complete adhesion of the two layers of the pericardium

a different result is usually observed. The muscular tissue beneath that membrane becomes imperfectly nourished and its activity impaired; its colour assumes a dirty brown, yellowish or straw-like tinge, its consistence is diminished, its fibres are easily separated and torn, and the whole mass becomes passively dilated in consequence of the pressure of the blood contained in the ventricles. This condition is more especially marked in chronic pericarditis, or when the exudation is of a purulent, hæmorrhagic, or tubercular character, or when the resulting abnormal tissue, which invests the heart, is tough and unyielding in its nature. The impulse, in such a case, will be evidently weaker than that of the organ in its healthy condition.

I have already stated that the impulse of the heart may be weak, and even absent, in persons in whom the most careful investigation can discover no trace of disease. Bearing this fact in mind in our examination of patients supposed to labour under cardiac affection, we find that the impulse may be abnormally weak.

1. In the second stage of pericarditis, when a large quantity of fluid has been exuded into the sac of the pericardium; the same result will, of course, follow upon the presence of an unusual quantity of fluid of any kind in this position. Whatever be the nature of the effusion, (serous, albuminous, sanguineous, or purulent,) the stroke of the heart becomes remarkably weak and irregular, and, in many cases, very variable in its position. When the patient lies upon his back, the heart, from its greater specific gravity, sinks towards the vertebral column, its anterior surface becomes covered by the

effusion, and its apex prevented from striking the chest-wall with its usual force. A partial paralysis of the muscular tissue of the heart from a physical or chemical change in its fibres also follows as a direct consequence of the imbibition of the surrounding fluid; the passive dilatation which ensues becomes, therefore, an additional cause of a weak and irregular impulse.

2. In atrophy of the heart, with or without dilatation of its cavities.

3. In passive dilatation.

4. In those diseases which exhaust the vital energies, and by their effect upon the constitution of the blood diminish its power of exciting the contraction of the muscular fibres of the heart; anæmia, chlorosis, scorbutus, typhus, &c.

5. In fatty degeneration of the heart, either in the form of fatty growth or of actual fatty conversion of the muscular fibres of the organ. In the former case the fat lies either upon the surface in a thick layer, or dips down at the same time between the fasciculi, producing by its pressure an atrophy of their substance. In the latter case the fibres lose their characteristic cross markings, and become filled with oily or fatty granules.

6. In emphysema of the left lung, causing the interposition of a quantity of dilated pulmonary tissue between the heart and the chest-wall, whereby the impulse of the apex is destroyed.

7. In persons whose chest-walls are covered with

loose, fatty, soft parts, either healthy or infiltrated with a quantity of serous fluid.

Extent of Impulse.—The extent of surface over which the heart's stroke may be felt or heard, depends upon the force of the impulse against the chest-wall, and the capability of the latter and of the parts surrounding the heart of conducting vibrations. The limits within which it may be perceived in health cannot be laid down with any rigorous precision. "The same man, according to the varying postures of his body, will alter the place and extent of the impulse. He stands up and makes it felt just when the apex strikes the chest, at a point between the fifth and sixth ribs, and not beyond. He leans forward and makes it felt both at this point and a little above it, and in the direction of the sternum. He reclines upon his back, and renders it almost or altogether imperceptible anywhere. He turns on his left side, and renders it more perceptible than ever, and in a somewhat larger and different space between the fifth and sixth ribs, and from thence more towards the mamma. Again, he turns on his right side, and again he renders the impulse almost or altogether imperceptible."* In practice it will be found:—

1. That the diseases already mentioned which increase the force of the impulse will also increase the extent over which that impulse is heard.

2. That all diseases which condense the surrounding lung parenchyma, or substitute a homogeneous conductor for the spongy lung, spread the effect of the

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* Dr. Latham, on the 'Diseases of the Heart.'

impulse to a greater extent than normal. These morbid conditions are pneumonic and tubercular consolidation and exudations of fluid in the left pleural cavity. Morbid growths in the chest lead to a similar result.

3. That malformations of the chest-wall, curvatures of the spine, or any cause which mechanically presses upon the heart and brings a larger surface of it than usual in direct contact with the parietes of the thorax, will cause a jarring movement of the heart capable of being heard almost over the entire surface of the chest.

Lastly, that the impulse of the healthy heart is often heard in every part of the chest in persons of an excitable or nervous temperament.

Fremissement Cattaire; Purring Tremor.—A peculiar thrilling vibratory motion, which, from its resemblance to the sensation experienced by the hand laid upon the back of a purring cat, has been designated as above, is sometimes felt in the præcordial region, and in that portion of the surface of the chest which corresponds to the position of the large vessels. This tremor may be often traced in many of the arteries of the body, the crural, carotid, temporal, and radial, and on applying a stethoscope over these vessels a loud systolic sound is found to be its constant accompaniment. Although the fremissement may sometimes arise solely from an abnormally low specific gravity of the blood, its cause, in the great majority of cases, undoubtedly resides in the presence of some obstruction to the circulation, either at one of the orifices of the heart or in some portion of the internal surface of the lining membrane of the arteries. Its distinctness and intensity will, therefore, depend upon—

1. The contractile power of the heart.
2. The quantity of blood expelled at every systole and driven through the narrowed channel or over the rough surface.
3. The nature, position, and extent of the obstruction.

When the heart becomes tranquil or its power of contraction weakened from any cause, the purring tremor diminishes in intensity and even disappears for a time, until some fresh excitement renews the systolic force upon which it depends. When the obstruction is at one of the orifices, the fremissement bears some relation, in its intensity, to the size of the channel through which the blood is driven in systole. Should the channel, however, become so narrowed, as to admit the passage of only a small stream of blood through it during the ventricular contraction, the fremissement becomes proportionally diminished, and, in some cases, completely annihilated. Hence it follows, that every diminution in the distinctness of the purring tremor cannot be always considered to be favorable to the patient.

The rubbing together of portions of lymph effused upon the opposed side of the pericardium may be often perceived in the palpation of the præcordial region, and the sensation which the friction gives to the hand is exactly similar, and not to be distinguished in character from the fremissement dependent upon endo-cardial causes. The internal is principally distinguished from the external fremissement by the former being exactly synchronous with the rhythm of the heart's systole and diastole, and by its evident connection with certain abnormal sounds detected by listening over the præcordial region. I have already mentioned the occurrence of a peculiar thrilling vibratory sensation in many of the arteries of the body, resembling in its character the fremissement cattaire perceived over the heart and its great vessels. This tremor is more especially observed in the radial artery in cases of an insufficient closure of the aortic orifice in diastole. In this disease an abnormal quantity of blood becomes accumulated in the eccentrically hypertrophied left ventricle; an abnormally large quantity is expelled during the systolic contraction (which is itself abnormally increased in force); a portion of the blood driven out in systole returns by regurgitation in diastole, and the pulse, as a consequence, acquires a rapid, short, vibrating, and thrilling character. The same fremissement may be also the result of the conduction along the course of the circulation of the vibrations of a purring tremor, which originates either in a stenosis of the arterial orifice, a roughened condition of the lining membrane of the aorta, or in a peculiar attenuated condition of blood.

LECTURE XIII.

PERCUSSION. AUSCULTATION.

As Percussion enables us to detect those conditions of the pericardium, heart and great vessels, which consist of abnormal deviations of positions and size, we should make ourselves familiar with the relations of these important parts in their state of health, in order to be capable of estimating the amount of lesion which they may have suffered from disease.

The devoted champion of Plessimetry, M. Piorry, with an exhibition of persevering research only equalled by his enthusiasm in the cause of Percussion, has laid down the rules by which, not only the entire circumference of the heart, but also the several parts which constitute that organ, may be discovered and traced upon the surface of the chest. "When the cavities of the right side have been determined, and the plessimeter has been carried still more inwardly, a marked resistance to the percuting finger and a greater dullness to the ear will be observed in the great majority of cases, sensations which are due to the position of the left ventricle."* I must confess that, although I have attempted with considerable patience to follow out the rules of this writer, and have witnessed his practice in the wards of the hospital at Paris to which he is attached, I have not been able to acquire that delicacy of touch and refinement of the sense of hearing so necessary to such exquisite diagnosis. I am rather of the opinion, so ably expressed by Dr. Latham in his remarks upon the healthy limits of the heart's sounds within the chest, that "no good ever comes from pretending to more precision than the thing itself admits of;" and am tempted to apply to the abnormally enthusiastic class of plessimetrists, the observation made, I think, by Dr. Williams, upon the writers who have described an endless variety of pulses, that "they exhibit a greater amount of sense at the ends of their fingers than they do elsewhere."

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* Piorry; 'Proc. Operat.,' p. 117.

The præcordial region, or the surface of the chest which corresponds to the portion of the heart uncovered by lung, is comprised between the cartilages of the fourth and sixth ribs and the mesian line of the sternum, and its upper boundary lies beneath the nipple of the left side. Its shape is usually described as resembling a lozenge; but we may rudely compare it to a triangle, whose sides are:—(1) the mid-line of the sternum which corresponds to the thin edge of the right lung; (2) a line which passes obliquely from the fourth sterno-costal articulation across the cartilages of the fifth and sixth ribs and marks the border of the left lung; and (3) a line which runs along the upper edge of the cartilage of the sixth rib. The area of this space varies with the recumbent and erect postures of the body, being less in the former than in the latter, and its extent depends also upon the degree to which the right and left lungs are inflated. Taking the mean of a number of healthy cases, we may estimate the area of the space uncovered by lung at 2½ to 3 inches, or if we measure from side to side, no diagonal of the figure can be found to exceed two inches in length. In this space, and only separated from the chest-wall by the pericardium and a quantity of loose cellular tissue, are situated a part of the right ventricle and that portion of the left which constitutes the apex of the heart. On gently percuting this region an absolute dullness is only obtained towards its central point, a spot which corresponds to the middle of the cartilage of the left fifth rib. Towards the periphery the sound is rather diminished than absent, in consequence, I presume, of the readiness with which the impulse of the percuting finger is communicated to the adjacent lungs. A more forcible impulse elicits the solid character of the subjacent heart. The parts surrounding the organ may be distinguished by the character of the sounds which they yield upon percussion. Thus percuting from the centre of the præcordial region upwards and laterally, we find the dull cardiac tone gradually merging into the full and perfect pulmonic sound, and below and laterally into the hollow tympanitic resonance of the stomach, and the duller sound of the left lobe of the liver, while the sense of resistance to the finger diminishes as the sonorous character of the sound increases. Careful examination enables us to trace, with tolerable accuracy, the outline of the heart, and more especially of that portion of it which is in direct contact with the thoracic wall, but I am fully convinced that no difference in the sounds or in the sensation of resistance can be detected between the two sides of the heart.

The aorta and pulmonary artery lying, during the act of inspiration, at a depth of nearly one inch beneath the sternum, modify, to a slight and almost inappreciable extent, the resonance obtained from the corresponding surface of the chest. The base of the heart whence these vessels originate, is usually detected on a level with the cartilages of the third ribs, and the arch of the aorta is found in the adult at the distance of nearly one inch from the upper edge of the sternum. Hence, with these data we may determine the extent of the space in which, from the position of the great vessels, a slight diminution of resonance may be expected. M. Piorry states that the breadth of this space, near the base of the heart where the aorta, pulmonary artery, and superior vena cava lie in juxtaposition, varies from 16—20 lines, while its breadth, when the aorta has escaped from the pericardium and is ascending alone behind the wall of the sternum, is only from 10—12 lines.

The morbid results obtained by percuting the præcordial region, are :—

1. Diminished extent of the region of dullness.
2. Increased ,, ,, ,,

1. In place of the usual dullness of sound and sensation of resistance to the percuting finger, we sometimes find an unusual resonance and absence of resistance from one or more of the following causes :—

(*a.*) Emphysema of the lung.
(*b.*) Atrophy of the heart.
(*c.*) Pneumatosis pericardii.
(*d.*) Hydro-pneumatosis pericardii.

The cases in which either air or air and fluid combined have been found in the cavity of the pericardium, are too rare to require any comment to be made upon them. Atrophy of the heart allows of a closer approximation of the right and left lungs in proportion to the diminution of the volume of the organ, and leads to an abnormal excess of sound in the præcordial region, with a diminished sensation of resistance to the percuting finger. Examples of this form of cardiac disease seldom occur to an extent sufficient to exhibit decided abnormal symptoms on percussion.

An emphysematous condition of the anterior edge of the left lung, is the most frequent cause of a diminished dullness in the præcordial region. The heart becomes not only partly displaced, but covered anteriorly by the dilated lung, and the area of the portion in contact with the thoracic parietes is abnormally lessened, at the same time that the sense of resistance is decreased.

2. An increased extent of the region of præcordial dullness, is a much more frequent condition. Its causes may be arranged in the following order:—

(*a.*) Effusion of fluid into the cavity of the pericardium:—

(*α.*) Blood—from rupture of the heart, or of an aneurism of the commencement of the aorta.

(*β.*) Serum—(hydrops pericardii) the result of a general anasarca of the serous cavities, due either to an obstruction to the current of the circulation or to a morbid condition of the blood, such as follows upon morbus Brightii, chlorosis, tubercular, exanthematous diseases, &c.

(γ.) Fibrinous, albuminous, purulent and hæmorrhagic exudations consequent upon pericarditis.

(δ.) Tubercular and cancerous developments, which are usually attended by serous effusion.

(*b.*) Abnormal enlargement of the circumference of the heart.

(*α.*) Hypertrophy—simple or eccentric.

(β.) Atrophy and dilatation of the cardiac walls.

(*c.*) Aneurismal dilatation of the commencement of the aorta.

(*d.*) Consolidation (tubercular or pneumonic) of the pulmonary tissue in the immediate neighbourhood of the heart and great vessels.

(*e.*) Morbid growths developed in the mediastinum, a collection of fluid in the cavity of the left pleura extending to the præcordial region.

It will be unnecessary to make any comment upon many of these morbid states; it is sufficient to know that such causes may produce the effect stated, and I shall only refer to the results exhibited by an abnormal collection of fluid in the pericardium, and by an enlarged condition of the heart.

Although a small quantity of a perfectly limpid, pale, yellowish green fluid is usually present in the pericardium after death, there is every reason to believe it to be the result, in great measure, of a condensation of the habitus which separates the opposed serous surfaces during life, and allows of their gliding noiselessly over each other during the unceasing movements of the contained organ. The normal quantity of this fluid is still undecided by physiologists. Corvisart estimated it at 6 ounces; Bouillaud at 1—2 ounces, while Rokitansky considers any amount beyond ½—1 ounce to be abnormal, and to constitute a case of hydrops pericardii. We are equally undecided upon the smallest quantity of fluid capable of producing an unusual extent of præcordial dullness, as the result must greatly depend upon the position of the lungs, with respect to the anterior surface of the heart, and also upon the presence or absence of previous pericardial adhesions.

A few ounces (4 or 5), have, in some cases, been detected by percussion, while double the amount has frequently escaped this method of investigation. Supposing no adhesions to be already present to unite the opposite sides of the pericardium, and that the elasticity of the sac has not been injured by any previous disease, the heart will evidently sink in the lighter fluid which surrounds it, whatever be the nature and source of the exudation. A moderate amount of effusion will be, therefore, indicated by an unusual degree of dullness towards the base of the heart, unless the presence of old adhesions, in that position, confine the fluid to the lower part of the sac. If the exudation be larger in amount and more persistent in duration, the elasticity of the pericardium becomes seriously impaired, and a distension of its parietes may follow to an almost incredible extent.

The sac assumes a pyramidal shape with its broad base resting upon the diaphragm, and its summit lying behind the upper part of the sternum. Its size depends, of course, upon the amount of fluid collected in its interior, and is, consequently, liable to endless variations. In an extreme case, related by M. Casimir Broussais, and quoted by Bouillaud, the dullness upon percussion extended perpendicularly from within two inches of the upper border of the sternum to the lower extremity of the suphoid cartilage, and in a horizontal direction from one nipple to the other.* In ordinary cases the distended pericardium pushes aside the anterior borders of the right and left lungs, and coming into contact with the chest-wall, causes the unusual præcordial dullness of percussion to be traced over the cartilages of the third and second ribs of the left side, frequently over the sternum, and, in some cases, as far as the insertion of the cartilages of the right upper ribs with that bone. The sensation of resistance is also considerably increased, and presents a remarkable contrast to the sensation derived from those parts in the normal state.

"When the quantity of fluid in the pericardium amounts to two pounds, the percussion sound is usually absolutely dull from the cartilage of the second rib of the left side to the lower border of the chest, and from the right edge of the middle bone of the sternum to the middle of the left side of the thoracic cavity. The resistance in this, as in extensive pleuritic exudations, is excessively marked." (Skoda.) From the remarks already made, it is evident that the effused fluid is capable of moving in the pericardial cavity, and of becoming collected in particular parts of the sac according to circumstances. Is it possible to trace this movement by percussion? Piorry, Maillot, Barth and Roger, and others, assure us that the superior level and lateral limits of the region of dullness upon percussion, vary with the position of the patient, and afford a means of distinguishing between an enlarged heart and an effusion of fluid into the sac. The opportunities I have had of watching the latter disease have failed to convince me of the truth of the statement. The elasticity of the pericardial structure is only impaired in extreme cases of distension, to such an extent as to allow of the outline of the sac to be altered by every posture which the patient is capable of assuming, and even here the exact boundaries of the effusion, in two different positions of the body, is excessively difficult to determine, in consequence of the proximity of the surrounding lung. Theoretically, the sac must be altered, in contour, by every change of position; but, placed as it is, within a bony and cartilaginous chest, and surrounded by a structure which is highly resonant on percussion, the alterations produced in its outline are practically more difficult and generally impossible to detect.

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* 'Traité Clinique des Maladies du Cœur,' vol. ii, p. 459.

The diagnosis capable of being afforded by percussion between an enlarged heart and a pericardial effusion, resides in its enabling us to observe the rapidity with which the latter, and the extreme slowness with which the former, disease are developed.

I have already said that the alterations of the size and shape of the heart, taken as a whole, and not of the parts which compress the organ, are revealed to us by percussion.

With respect to increase of size, I know nothing more difficult, in the investigation of cardiac disease, than the exact determination of the point at which an augmentation of volume commences, for no definite rules can be laid down which allow us to fix unerringly the limits of the healthy heart, and to assert when dilatation, active or passive, has begun. An examination, by percuting the præcordial region during life, must be consequently attended with considerable uncertainty as to the result in the early stages of such diseases; the difficulties of which are increased by the fact of the volume of the heart, in its normal state, being known to vary with the age, sex, temperament, stature, capacity of chest, and mode of life of the person under investigation. To which may be added the possibility of an emphysematous condition of those portions of the lungs which extend over the anterior surface of the organ, and mask its normal or abnormal outlines.

One or both sides of the heart may become the seat of enlargement, and present every degree of increase, from that amount which is scarcely worthy of mention to those enormous developments which sometimes occupy a very large portion of the thoracic cavity. Every one who has attended the post-mortem examinations of a hospital must have had ample opportunities of witnessing enlarged hearts. Bouillaud relates an instance in which the heart considerably exceeded three times the size of the fist; another, in which the heart of a girl, æt. 7, occupied about one third of the anterior part of the thoracic cavity. "I have met with a heart which weighed 40 oz.; its circumference round the base was $14\frac{1}{2}$ inches; its length from the arterial orifices along the septum to the base $8\frac{1}{2}$ inches; thickness of the left ventricle 1 to 4 inches, near the columniæ corneæ 1 to 5 inches."* It is evident that an increased thickness and weight must be associated, in such cases, with an augmented volume. The walls of the left ventricle, in place of averaging five lines, increase to 1—2 inches in thickness, while those of the right ventricle reach to $\frac{1}{2}$ — $\frac{3}{4}$ of an inch. The weight, increasing in proportion to the augmented volume, ranges from 10 to 40 oz., and upwards. An important change takes place also in these cases of enlarged heart. The organ sinks from its increased weight, and lies more transversally in the chest than usual, with its base behind the sternum and its apex to the left of its ordinary position, and, resting upon the diaphragm with a large flat surface, forcibly depresses it into the epigastrium. Changes of such import in the volume and figure of the heart, and the corresponding effects upon the surrounding parts, will evidently be detected by percussion. The lungs become separated from one another, and pushed aside by the gradual advance of the abnormal mass. An unusually large portion of the organ lies directly under the thoracic wall, and leads to an increased region of præcordial dullness and augmented sensation of resistance to the percuting finger.

The remarks hitherto made have principally referred to those cases of enlargement which are due to eccentric hypertrophy (active dilatation) of the heart. The results will be the same whatever be the cause of the increase in volume. We are told by some authorities, that the sensation of resistance is not so marked in passive as in active dilatation; perhaps the statement may be true. I have no faith in its correctness; but whatever be the fact on this point, there is no doubt that the extent of præcordial dullness is always proportional to the increased size of the organ, and the displacement produced by it of the neighbouring organs. Simple dilatation is the frequent and constant result of disease of the left side of the heart, or of some obstruction to the pulmonary circulation. It may be found in all degrees of development; but, in the majority of cases, is usually associated with hypertrophy, and constitutes the form called active dilatation. The cases are very rare in which the entire organ presents a pure example of passive dilatation.

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* Dr. C. J. Williams, 'Diseases of the Chest,' p. 248.

Auscultation.—On placing the stethoscope over the apex of the heart, two sounds become distinctly audible, following each other with great rapidity, and respectively synchronous with the contraction and dilatation of the ventricles. The systolic, or first sound, and the diastolic, or second sound, may be imitated by articulating the syllables lubb, dŭp (Dr. C. J. Williams). If we listen over the middle bone of the sternum and on its left edge, (a spot which very nearly corresponds with the position of the arterial valves,) similar sounds become audible, with this difference, that the first is less loud and prolonged than the systolic sound at the apex; while the second is clearer, sharper, and more distinctly defined than the diastolic sound in that position.—The first is synchronous, or nearly so, with the pulse of the radial artery and with the stroke of the heart against the chest-wall; the second with the depression of the pulse and retreat of the heart from the chest-wall. A little observation will also show that the sounds occur in a certain order of succession—the rhythm of the heart—which is as follows:—

1st sound—small silence;—2d sound—long silence, and is commenced and terminated in the interval of time between two successive pulses, in a period equal to $\frac{1}{70}$th of a minute (if we suppose the pulse to beat at the rate of 70 per minute).

As the duration of the small silence cannot exceed $\frac{1}{420}$th part of a minute, or $\frac{1}{7}$th of a second,—a period of time almost incapable of being appreciated by the ear, the 2d must appear to follow immediately and without any measurable interval upon the 1st sound. The time from the commencement of one systolic to the beginning of the next systolic sound (during which one ventricular contraction, one ventricular dilatation, and one ventricular rest occur) is, consequently, divisible by the ear in a period of sound and a period of silence. What are the relative lengths of these two periods? Laennec estimated the time occupied by the two sounds, and the intervening rest between them, to be rather more than three times the period of silence; Dr. Williams considers their ratio to be nearer that of two to one.

"The two sounds with the intermediate silences constitutes a beat or rhythm, and to each beat corresponds an arterial pulsation. The result is, therefore, a kind of triple measure (*mesure à trois temps*), of which the first sound occupies a little less than a third of the time, the short silence and the second sound each a sixth, and the long silence a little more than a third."* Let us for a moment estimate the durations of these periods; and, according to the above data, if we suppose the pulse to beat at the rate of 70 times per minute:—

Duration of first sound $= \frac{1}{3} \cdot \frac{1}{70} = \frac{1}{210}$th of one minute nearly
,, short silence $= \frac{1}{6} \cdot \frac{1}{70} = \frac{1}{420}$,, ,, ,,
,, second sound $= \frac{1}{6} \cdot \frac{1}{70} = \frac{1}{420}$,, ,, ,,
,, long silence $= \frac{1}{3} \cdot \frac{1}{70} = \frac{1}{210}$,, ,, ,,

Periods of time so small are evidently beyond the power of the unaided ear to estimate; and a little reflection on the mode usually adopted by writers of measuring the relative lengths of the periods of sound and silence, will lead us to doubt the minute results at which they have arrived. Let any one apply a stethoscope over the heart of a healthy individual, and attempt to form any accurate division of the time occupied by sound and silence,—a time, which, on the average, rarely exceeds $\frac{1}{70}$th part of a minute, *i. e.*, less than a second,—I am certain that he will come to the conclusion that he cannot tell whether Lacnnee, Barth, or Williams are correct, who respectively state the period of sound to be $\frac{3}{4}$, $\frac{2}{3}$ and $\frac{1}{2}$ of the interval between two consecutive pulses. Observe the excessively small difference between the estimates formed by Laennee and Barth, and say whether such delicate differences can be distinguished by the unaided ear. Thus, as according to Laennec, the period of sound occupies $\frac{3}{4}$ of the time of a whole pulse, or $\frac{3}{4} \cdot \frac{1}{70}$ of a minute, and, according to Barth and Roger, only $\frac{2}{3}$ of a pulse, or $\frac{2}{3} \cdot \frac{1}{70}$ of a minute, the difference between their estimates $= (\frac{3}{4} - \frac{2}{3}) \frac{1}{70} = \frac{1}{12} \cdot \frac{1}{70} = \frac{1}{840}$th part of a minute, or $\frac{60}{840} = \frac{1}{14}$th part of one second,—a period totally inappreciable by the unassisted ear; and the same remark applies to the other estimates. From these considerations we might, therefore, despair of obtaining an accurate measurement of these periods; but we have fortunately a work lately brought to our notice, in which the subject has been treated scientifically. In the elaborate treatise upon Hæmodynamics, by Professor Volkmann, to which I have already referred, we find a series of careful experiments described to determine the point in question. Two half-second pendulums were obtained, the durations of whose vibrations could be increased or diminished by moveable weights attached to their stems. While listening to the action of the heart of a healthy individual at rest, the Professor so arranged that the duration of the vibration of one pendulum should coincide with the period of sound, and that of the second pendulum with the period of silence. By this means the times of these periods were directly measured. In order, however, to test the accuracy of his results, he compared the duration of the interval between two successive beats of the radial artery with the sum of the durations of the periods of sound and silence found in the experiment, and by the smallness of the discrepancy in the results, proved the correctness of this method of investigation. He gives the following as an instance:—

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* Barth and Roger.

N. N— æt. 34 years,—Pulse 84 in a minute.

By experiment—

Period of sound = 0·3750 seconds.
Period of silence = 0·3798 „

Interval of one pulse = 0·7548 „

By calculation—

Interval of one pulse $= \frac{1}{84}$th part of one minute
$= 0{\cdot}7140$ seconds.

Hence, the discrepancy between experiment and calculation amounts only to 0·0408 seconds, a period of time totally inappreciable by the ear.

We also deduce from the experiment the fact, that the periods of sound and silence are nearly equal, being in the ratio of nearly 99 to 100. The result differs from the statement of all preceding authors who have considered the duration of the period of sound to be always greater than that of silence. It differs least, however, from the estimate formed by Dr. C. J. Williams whose labours have thrown so much light upon the entensive field of the physiology and pathology of the heart.

LECTURE XIV.

AUSCULTATION.

Continuing our observations upon the character and nature of the normal sounds of the heart, we have now to consider the causes to which they may be correctly referred—a subject of no small difficulty, and considered by many able physiologists to be still *sub judice.* As the object and limits of this course preclude any lengthened detail of the numerous theories which have been advanced by authors, English, French, and German, upon this subject, I shall restrict myself to a simple statement of those which appear to be the most important, and only dwell upon the view which accords, in my mind, the most with the facts observed in the healthy and diseased conditions of the heart and its great vessels. And at the outset, let us observe, the actions which take place in the heart at the time when the sounds are produced. During the systolic, or first sound, the apex of the organ strikes with considerable force against the chest wall, the ventricles contract with an energy capable of propelling their contents to the most distant parts of the body, the curtains of the mitral and tricuspid valves are suddenly stretched by the action of the papillary muscles and tendons, and by the impulse of the blood on their lower surfaces compelled to close the auriculo-ventricular openings, the stream of the circulation is directed towards the origins of the aorta and pulmonary artery, driving the semilunar valves against the sides of those vessels, and lastly, a general molecular collision takes place of the fluid contents of the ventricular cavities. And all these actions are commenced and terminated in a period of time, which, on the average, does not exceed the $\frac{1}{140}$th part of one minute, *i. e.* $\frac{3}{7}$ths, or less than $\frac{1}{2}$ of a second (supposing the pulse to beat at the rate of 70 times per minute). During the period of the diastolic sound, the blood insinuates itself behind the semilunar valves, and by its recoil upon their upper surfaces suddenly compels them to close the orifices of the aorta and pulmonary artery, while at the same moment, the contents of the auricles commence to be discharged into the ventricles through the auriculo-ventricular openings. The duration of the diastolic sound is so short as to appear almost instantaneous, and rarely exceeds $\frac{1}{210}$th of one minute, or $\frac{2}{7}$ths of one second.

Observing then, the number of contemporaneous actions proceeding during the contraction of the ventricles, each capable, more or less, of producing sound, we might reasonably imagine the systolic sound to be the result of their combination. Such, however, has not been the opinions of the majority of writers upon the Physiology of the Heart. Thus, in our own country, we find Dr. C. J. Williams attributing the systolic sound to the tension of the ventricular muscular fibres consequent upon their contraction. Dr. Hope describes it as "a loud, smart sound, produced by the abstract act of sudden jerking extension of the already braced muscular fibres at the moment that the auricular valves close." My late father said, "I believe, that the first sound is caused by the point of the heart striking against the ribs."[*] The Dublin Committee came to the opinion, that the sound results partly from muscular contraction, and partly from the friction of the blood in its passage over the walls of the ventricles. Dr. Blakiston refers it to a friction of the muscular fibres *inter se* during the contractions of the chambers. Dr. Billing believes it to be due to the sudden tension of the auriculo-ventricular valves—an opinion in which he accords with Bouillaud and Rouanet. Cruveilhier attributes the sound to the flapping back of the semilunar valves against the sides of the large vessels during the ventricular systole; an opinion in which he is supported by our eminent physiologist, Dr. Carpenter, who states, however, that "observations do not entitle us to deny the participation of the muscular contraction and the movement of the blood over the ventricular walls in the production of the first sound."[†] Majendie maintains the view of the sound being caused by the stroke of the heart against the ribs. The distinguished German physiologist, Valentin, maintains the theory entertained by Bouillaud and Rouanet, and adds, in confirmation of his view, the statement, that if drops of water fall from a height upon a portion of intestine fully distended with water, the sound heard by means of a stethoscope placed upon the intestine, closely resembles the systolic sound of the heart.* You will find the *pros* and *cons* of these several theories fully given by MM. Barth and Roger; and even more lucidly in the admirable lectures lately published by Dr. Bellingham, upon the 'Diseases of the Heart.' This author very ably maintains the view, that the systolic sound is the result of the friction of the blood against the parietes of the orifices of the organs, a theory also entertained by Gendrin and Pigeaux.† MM. Barth and Roger refer the sound to the complexity of causes described at the commencement of this Lecture, and agree in the main with the theory advanced by Professor Skoda, to which I shall immediately refer.

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[*] Dr. Thomas Davies, 'Lectures on the Diseases of the Lungs and Heart,' p. 388.

[†] 'Principles of Human Physiology,' p. 398.

The diastolic sound is admitted on all hands to be much more simple in its origin. Almost all writers of the present day have agreed to refer it to the sudden tension of the semilunar valves, caused by the recoil of the columns of blood in the aorta and pulmonary artery upon the upper surfaces of those delicate folds of membrane. The simplest and best proof of the correctness of this view is afforded by the fact, that any injury to the valves in the vivisections of animals, or as the consequence of disease, entirely abolishes the sound, and replaces it by a murmur of a totally different character.

Where so many circumstances coexist capable of producing vibration, it is difficult to decide upon the predominating cause of the first sound. The impulse of a solid body, like the heart against the ribs, must evidently be attended by the evolution of a distinct sound. We have only to tap gently with the finger against the internal surface of the chest of a subject under a *post-mortem* examination, and listen at the same time with a stethoscope over the spot percuted, to be convinced that the stroke of the heart plays an important part in the production of the systolic sound. To what extent sounds generated within the cavities of the ventricles by the molecular collision of the contained blood, by the friction of that fluid over the surfaces of the endocardium and valves, and by the tension of the valvular apparatus, may be capable of being conducted across the muscular parietes of the heart to the thoracic wall, we are not fully in a position to decide. A very large proportion of the vibrations must be undoubtedly stifled, and lost in transitu; still we have every reason to believe, that a portion is communicated to the chest-wall, and heard to mingle with the sound produced by the heart's impulse. The valuable experiments made by Dr. C. J. Williams, undoubtedly establish the existence of a *bruit musculaire*, or sound, dependent upon the sudden contraction and state of tension into which the fibres of the ventricles are thrown during systole. We have seen that this action is commenced and terminated usually within the short period of $\frac{1}{140}$th part of a minute, and its force we have calculated in a former lecture, and shown to be of considerable amount. Hence, we can have no difficulty in concluding, that a contraction so rapid and forcible must be attended with the evolution of sound; and, if an example of the kind is required to confirm us in our belief, we have only to apply the stethoscope over the adductor muscle of the thumb, and listen to the sound which accompanies the vigorous movements of that muscle.

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* Valentin, 'Lehrbuch d. Physiol. der Menschen.

† Dr. Bellingham, 'Lectures of the Diseases of the Heart.' Medical Gazette, 1850.

As both sides of the heart participate in the production of the normal sounds of systole and diastole; and as disease, involving structural changes, generally confines itself to the valves and orifices of the left side, we might *à priori* expect to find, in some cases, a want of accord in the sounds of the right and left sides of the organ. Clinical experience fully corroborates this view. And from the observed results of health and disease, Professor Skoda constructs a theory, which, with one or two exceptions, appears to offer the best explanation of the origin of the sounds.

This skilful auscultator considers—

(1.) That the right and left ventricles, the aorta and pulmonary artery, combine in the formation of these sounds.

(2.) That of the systolic sound, one portion is due to the ventricles, and the remainder to the origin of the great vessels.

(3.) That in a normal condition of the heart, the synchroneity of the causes which produce sound in the heart and large vessels, causes the two portions to be blended together into one sound.

(4.) That in disease a separation of the two portions respectively due to the right and left side of the heart becomes evident to the ear. Thus in organic disease of the aortic valves, their first and second sounds are replaced by a double murmur; but normal systolic and diastolic sounds may be heard over the valves of the pulmonary artery; and a normal systolic sound (I imagine due to the stroke of the heart against the ribs) over the apex of the organ.

(5.) That the ventricular first sound is due to the stroke of the apex of the heart against the chest-wall, the sudden tension of the auriculo-ventricular valves, and the impulse of the blood upon their tense surfaces during the closure of the orifices to which they correspond. (To these causes, I believe, the *bruit musculaire* should be added.)

(6.) That the arterial first sound results from the suddenly increased tension of the coats of the aorta and pulmonary artery, produced by the shock of the blood impelled upon them.

(7.) That the ventricular second sound is either the second sound propagated from the semilunar valves, or the result of the shock of blood against the ventricular walls, and the sudden disengagement of the apex of the heart from the opposed pericardium.

(8.) That the arterial second sound proceeds from the vibrations of the valves of the aorta and pulmonary artery, rendered tense by the sudden recoil of the blood upon their upper surfaces.

Complex as this theory may appear, I believe, that its main points can be substantiated. I have no practical knowledge, however, of a ventricular second sound *per se,* and I cannot acknowledge the causes stated as capable of producing it. The force with which the blood passes into the ventricles from the auricles is confessed by all practical writers, who are aware of the rarity of a direct mitral bruit, to be too small to produce sound; and the statement of sound being the result of the sudden disengagement of the visceral from the parietal layer of the pericardium, rests upon evidence too shallow to be for one moment admitted. Perhaps, the molecular collision of the blood during its recoil from the suddenly-closed semilunar valves, has been too much neglected in the above theory of the second sound. Dr. Hughes happily illustrates its production by the recoil-noise which is heard when the closure of a stop-cock suddenly arrests the flow of water through a pipe. Every one must be familiar with the sound. "This agitation, and this noise, are clearly the consequence of the vibrations excited in the fluid by the sudden arrest of its onward progress; which vibrations are communicated to the solid walls of the pipe, and thus become obvious to the eye as well as to the ear."*

A similar recoil takes place in the aorta and pulmonary artery, and serves to explain the occurrence of one portion of the diastolic sound. Changes in the muscular tissue of the ventricular walls (hypertrophy, atrophy, or fatty degeneration,) can only affect the systolic sound in its intensity and loudness, and occasionally in its sharpness and clearness, inasmuch, as upon the degree of the heart's energy of contraction will depend the force of the impulse, the *bruit musculaire,* and the molecular collision of the contained blood. Changes in the valvular apparatus and orifices of the heart, introduce, however, a class of murmurs which may replace one, or both, of the normal sounds at the apex or base of the heart. Hence the following practical points may be deduced:—

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* Dr. Hughes, 'A Clinical Introduction to the Practice of Auscultation,' &c., p. 185.

(1.) That a clear and distinct systolic sound heard over the position of the heart's apex, indicates the perfect closure of the auriculo-ventricular orifices. The same sound audible over the bases of the organ, proves the absence of any impediment to the free passage of the blood through the orifices of the aorta and pulmonary artery.

(2.) That a clear and distinct diastolic sound heard over the position of the semilunar valves, testifies to the perfect manner in which the orifices of the larger vessels are closed by those delicate folds of membrane.

Admitting the systolic and diastolic sounds to originate in the mode described, we are now enabled to account for those alterations of loudness, intensity, and rhythm, which they frequently exhibit. And with respect to the loudness of the systolic sound, we find the principal circumstances which modify its intensity to be—

(1.) The force of the ventricular contraction.

(2.) The capacity of the ventricles, and consequently the quantity of blood, usually expelled at every systole.

(3.) The elasticity of the coats of the aorta and pulmonary artery.

(4.) The nature of the medium through which the sounds are conveyed to the ear.

The greater the force with which the ventricular action is effected, and the larger the quantity of blood expelled at every systole, the greater will be the tension of the auriculo-ventricular valves, the more violent the shock of the blood against their surfaces, the stronger the stroke of the apex against the thoracic wall, the more powerful the friction of the blood in its passage through the orifices of the large vessels, the louder the systolic sound, and the greater the extent of the surface of the chest over which it may be heard. The shock of the impulse is sometimes so strong as to impart a kind of metallic kling (metallic cliquetis) to the first sound, very similar to the tone heard when one hand is placed over the ear, and its back struck by a finger of the other hand.

Again, as the second sound chiefly arises from the contractile force of the coats of the aorta and pulmonary artery, any cause which diminishes that force will produce a proportional diminution in the intensity of the sound, and *vice versâ*. Thus an unusual intensity of the second sound of the pulmonary artery always accompanies any disease of the mitral orifice, which impedes the flow of the blood from the left auricle into the left ventricle, or allows of the regurgitation of blood from the one cavity into the other. In consequence of the obstruction, the left auricle, the four pulmonary veins, and the system of the pulmonary artery, become inordinately filled with blood. The right side of the heart becoming hypertrophied and dilated, propels its contents with an unusual amount of force into the pulmonary artery. The coats of that vessel yielding to the increased force, react more strongly upon the contained blood, and the consequently increased recoil upon the semilunar valves produces an unusual loudness in the character of the diastolic sound.

The condition of the parts which surround and overlap the heart must greatly influence the distinctness and fulness with which the normal sounds reach the ear, and the distance to which they may be heard from their points of origin. A heart unusually covered by emphysematous lung, or removed to a distance from the chest-wall by the presence of an accumulation of fluid in the cavity of the pericardium or pleura, must have its sounds considerably diminished in intensity, and frequently displaced; while on the other hand, a consolidated condition of the surrounding pulmonary parenchyma (resulting from external compression or deposition of solid matter within the cells) must favour the conduction of the sounds over a large extent of chest-wall, and render them much more sensible to the ear.

The rhythm of the heart also deserves a few words of comment. I have already stated the order of succession in the healthy organ to be—

First sound—Short silence;
Second sound—Long silence:

and I have, in a former Lecture, given the relative and absolute durations of the several parts of this rhythm. The alterations observed, are usually confined to the systolic sound and the long silence. Thus an obstruction at the aortic orifice, which is insufficient to produce a murmur, or an inordinate accumulation of blood in the ventricular cavities, requiring an increased period of time for its expulsion—in a word, any cause capable of prolonging the ventricular systole, whether residing in the muscular tissue of the organ, or in the blood passing through its cavities, must tend to lengthen the systolic sound. The duration of the first sound is, of course, diminished when the rapidity of the pulse is increased. The short silence and diastolic sound occupy excessively small periods of time in health, and their deviations in disease are too small to be appreciated or estimated by the ear. The causes which prolong the duration of the long silence are,—either an infrequency in the action of the heart, or some delay in the filling of the ventricles with blood, from an obstruction at the auriculo-ventricular orifices. M. Barth cites an instance where a decoction of digitalis was administered by mistake to a young girl as an enema, instead of being employed as a warm application to the præcordial region. The beats of the heart fell to twenty-five per minute, and the principal alteration of the rhythm was shown in a marked prolongation of the period of silence. If we suppose the duration of the ventricular contraction to have been but slightly affected by the diminution of the number of the ventricular contractions per minute, the following may be considered as a kind of approximation to the length of time occupied by the long silence.

When the heart beats . . 75 times per minute,
The duration of one pulse . . $= \frac{1}{75}$th of a minute,
,, of the period of sound $= \frac{1}{150}$th ,, nearly.

When the heart beats . . 25 times per minute,
The duration of one pulse . . $= \frac{1}{25}$th ,,

Hence, on the supposition made above—

The duration of the prolonged silence = time of one prolonged pulse,
— time occupied by period of sound,
$= \frac{1}{25} - \frac{1}{150} = \frac{1}{30}$th of a minute nearly,
= 2 seconds nearly.

A silence of such a length must be marked indeed; but we cannot suppose it to be prolonged in cases of unusually slow pulse, without the period of sound exhibiting at the same time some increase of duration. Dr. C. J. Williams mentions the case of a gentleman whose pulse beats only twenty-eight in a minute, and systolic sound occupied nearly a second of time. The long silence here must have been prolonged beyond one second.

An irregularity in the succession of the ventricular contractions—an intermittence—is a well-known form of abnormal rhythm. The heart contracts rapidly for two or more times, and pauses for a short period to recommence the same or a similar order of succession. The irregularity appears in some cases to follow a constant law, and to have some method in it. In other instances the ear can only discover an erratic succession of sounds, destitute of every kind of order.

The ventricular contractions, though regular in succession, may be irregular in strength; but more commonly the two forms of irregularity are combined. Sometimes the abnormal rhythm appears to be periodical, and to occur at fixed intervals. Very often the pulse and the beats felt at the heart fail to agree. Some of the weak contractions do not reach the radial artery, and the pulse appears slower than the heart. Hence, in all cases of intermittence we should compare the pulse at the wrist with the heart, for the former may be irregular, while the latter is natural, and vice versâ.

An unusual number of sounds during the period of a pulse is another, but very rare, form of altered rhythm. Sometimes one sound is absent (usually the second according to MM. Barth and Roger), either from the original feebleness of the recoil action of the large vessels, (most probably dependent upon a feeble impulsion of the blood into them from the ventricular cavities,) or from the diastolic being masked by the prolongation of the systolic sound. In some cases a triple has been heard in place of the double sound, a result supposed to proceed from the valves of the aorta and pulmonary artery failing to fall synchronously with each other, or from one of the valves of either orifice falling into its place a little after its companions, and producing a supplementary second sound. Cases are even mentioned in which four sounds have been heard in the interval between two successive ventricular contractions. A phenomenon so unusual can be only ascribed to a most remarkable absence of concord in the action of the two sides of the heart. An utter want of synchronous action between the right and left sides may appear incredible, but the following quotation from

Volkmann may serve to illustrate this curious condition of things :—"When we open the chest of an animal which has been recently killed, the movements of the heart, which are at first regular, are soon seen to lose all order of succession. The auricles pulsate more frequently than the ventricles, and contracting 5, 10, and even 100 times in the period of one ventricular contraction. At a still later period, the synchronism of the left and right sides of the heart disappears. The one pulsates more frequently than the other, or the one pulsates while the other has lost all capability of independent motion."

I have in a former Lecture described the arrangement of the nervous system of the heart,—the number of nervous ganglia which lie scattered through its substance, but connected together and rendered capable of acting in unison by means of connecting filaments. Circumstances difficult to comprehend may disturb the unanimity of these ganglia, and produce the want of synchronism to which the abnormal multiplication of the heart's sounds has been referred. A greater amount of information than we possess is still requisite to enable us to decide upon the bearing of an irregular intermittence upon the duration of life; I mean that form of intermittence which is not associated with any cognizable disease of the valves or orifices of the heart.

Although intermittence in the heart's action is, by no means, a favorable phenomenon, yet there are many individuals in the apparent enjoyment of good health who have had an intermittent pulse for years, and without at any time feeling any ill consequences from this anomalous action of their hearts. These are cases of an intermittence in the force and number of the beats, and independent of all morbid murmurs, occurring at the orifices of the organ. I have met with a few examples of that kind, but I have no experience of the altered rhythm, shown by an unusual number of sounds taking place in one pulse. Skoda very ably sums up in a few words all that may be said on the importance of intermittence.—"Abnormal rhythm of the heart may be undoubtedly the frequent result of an organic change in the organ; but it is also true that the greatest irregularity of rhythm may prevail in hearts which are apparently perfectly normal in structure, while, at the same time, there exists no organic change of the heart and its valves, in which a perfectly regular rhythm cannot occur. Finally, we cannot infer from the existence of an irregular rhythm, however excessive, that an organic disease of the heart is necessarily present."*

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* Skoda.—Abhandlung über Percussion und Auscultation.

LECTURE XV.

AUSCULTATION.

We proceed to the consideration of a highly important class of sounds,—the abnormal murmurs of the præcordial region,—which, according as they originate between the layers of the pericardium or within the cavities of the heart, are termed:

$$\left.\begin{array}{l}\text{1. The exocardial}\\ \text{2. The endocardial}\end{array}\right\}\text{murmurs.}$$

1. *The exocardial or pericardial murmur.*

I have already described the pericardium to be a fibro-serous bag, the serous lining of which is reflected over and covers nearly two inches of the aorta and pulmonary artery, and the entire surface of the heart. During the contractions and dilatations of the several parts of the organs in its healthy state, no sound can be heard to proceed from the gliding of the opposed serous surfaces over each other; nothing can be discovered in the præcordial region but the normal sounds of systole and diastole. This silence of the heart's movements in its investing sac continues so long as the smoothness of the pericardial lining remains intact, but is immediately replaced by a distinct friction sound whenever the epithelium has been removed, and the surface of the serous membrane become roughened by disease.

A variety of morbid circumstances, such as the deposition of tubercular or cancerous matter within the tissue, or upon the free surface of the pericardium; the escape of blood into the sac, in consequence of an aneurismal rupture of the commencement of the aorta, &c., may give rise to physical conditions capable of producing a friction sound; but, for all practical purposes, we may confine our attention to pericarditis, as the most frequent and important cause of this remarkable murmur. The opportunities of examining patients at the earliest stage of the disease are very rare. I mean at the stage of simple inflammatory injection, when the inner surface of the pericardium has lost its smooth, glistening, and semi-transparent aspect from an infiltrated condition of its tissue, and presents the appearance of a delicate red membrane traversed by prominent streaks of the same colour ramifying over it in all directions. I have had no experience of the auscultatory symptoms of the disease at this early period of its existence, and, although I should, *à priori*, expect the production of some sound from surfaces deficient in lubricating fluid, and traversed by a multitude of injected vessels, yet we are told, as the results of some experiments, made by Dr. C. J. Williams before the British Association, that no murmur can proceed from the inflammatory injection, unless an effusion of blood or lymph be, at the same time, present under the pericardial lining. The duration of this early stage is usually too brief for observation, as an exudation of delicate flakes of coagulable lymph follows almost immediately upon the establishment of the vascular injection, and produces the physical conditions which are essential to the formation of a friction sound. Should the inflammation terminate with great rapidity, and the fluid effused be sufficient in quantity to redissolve the solid matter which had been exuded upon the inflamed surface, the entire product of the disease may be rapidly removed by absorption, and the serous membrane of either or both sides of the pericardium become restored, with the exception of some loss of transparency, to their original condition. With the removal of the abnormal contents of the pericardium will, of course, follow a cessation, and total disappearance of the friction sound. A result so favorable is not the usual termination of the disease, the course of which may be thus briefly sketched. A sero-albuminous fluid, rich in plastic lymph, is exuded from either surface of the pericardium, visceral or parietal, or from both simultaneously, in a quantity proportional to the intensity of the inflammation. By the rapid coagulation ensuing, flakes of fibrin become separated and thrown down upon the sides of the sac, but accumulated in greater abundance upon the serous portion investing the base and upper part of the heart. In many cases the entire organ becomes covered with the plastic lymph, but, as before stated, to a greater amount always towards the origin of the great vessels. A friction sound is the direct consequence of this condition, the duration and character of which will depend upon the changes taking place in the false membrane exuded. Thus, a rapid union of the opposed surfaces and a commencing organisation of the intermediate morbid deposit, bring the murmur to a speedy and permanent termination. The data are not yet known, which enable us to state the period of time in which this favorable result may be obtained. According to Zehetmayer, the exuded fibrin may exhibit symptoms of an organising process within forty-eight hours from the time of its deposition upon the pericardial surface, and present distinct traces of the presence of areolar tissue within a period of fourteen days. The fluid part of the exudation, however, frequently accumulates to an amount sufficient to separate the roughened surfaces, and thereby causes a cessation of the friction sound, until an absorption of the fluid brings the two surfaces again into contact, and restores, for a time, the conditions requisite to the production of the murmur. The character of the sound depends upon the nature of the rubbing surfaces; their softness, hardness, thickness or thinness, and also upon the force with which the movements of the heart are effected. It may be smooth and similar to the sound made by gently passing the finger over silk, or it may be harsh, grating and unpleasant to the ear, in consequence of a partial conversion of the morbid exudation into fibro-cartilage, or from the deposit of calcareous particles within it. The terms *frottement*, or simple rubbing; *craquement de cuir*, or the creaking of new leather; *râclement*, or grating sound, have been assigned by French writers to the different varieties of the murmur. In many cases the friction is sufficiently intense to communicate a distinct sensation of thrill; a *fremissement cattaire*, or purring tremor to the hand placed upon the præcordial region. The murmur may be heard over the whole or any portion of that space; it usually accompanies the systole and diastole, but with a greater intensity the former than the latter period of the heart's action, and when distinctly established is the sure indication of the presence of a quantity of solid matter in the pericardium unattended by the exudation of any considerable amount of fluid.

I have made use of the expression "distinctly established," because a pericardial may be, and has been, frequently mistaken for, and confounded with, an endocardial murmur. The latter usually proceeds from the friction of the blood through an abnormally narrowed channel, and presents, very frequently, a similarity so close to the sound of the pericardial friction, as to be with difficulty distinguished from it. Both sounds may occur during systole and diastole, both may produce a fremissement cattaire, and both may have a soft and smooth or a harsh and grating character. There are, indeed, many cases in which it is almost impossible to determine, from the sound itself, whether its origin is endo- or pericardial. Dr. Hughes, in his excellent work upon Auscultation, says:—"The pericardial occasionally so closely approximates in character and situation to the valvular murmur; it appears, indeed, as regards the sound, so perfectly identical with it, that I hesitate before I give an opinion, or I feel compelled to acknowledge my inability to form one;"—adding, however, that "these are exceptional cases, which constitute, very rare exceptions to a very general rule."—But we may naturally ask, what are the characteristic and essential differences between the two classes of sounds; what are the signs which enable us to assert that one sound is exocardial and another endocardial? We are told in almost every work which treats upon the subject, that the pericardial murmur appears more superficial and nearer to the ear of the auscultator than the abnormal murmurs which originate within the cavity of the heart;

and that a diagnosis is thus presented between them. Let me show you, by a very simple experiment, the degree of reliance to be placed upon the statement. I have here a large cylinder made of gutta percha, across the interior of which I have laid a thin stick of wood. Now let any one, by means of a stethoscope applied to the exterior of the cylinder, and over the spot which corresponds to one end of the stick, listen to the sounds made by scratching different parts of it with a pin, and attempt to discover, by the more or less superficial character of the sounds, the relative distances from the ear at which they are produced. He will find that strong friction, made at a spot at some distance from the end of the stethoscope, will produce a sound apparently more superficial and nearer to him than one made at a spot actually nearer but with less force, and that the relative distances of the points of friction cannot be invariably determined by the intensity of the sounds which are propagated from them to the ear. In the common experience of daily life, we are constantly taking loudness as a measure of the distance of sounding bodies, and when their exact position is unseen, we naturally fall into the idea of those being the nearest to us which appear to be the loudest. Nothing is, however, more deceitful and likely to lead us into error. The ventriloquist plays upon this peculiarity of our minds, and by simply varying the loudness of his imitation-sounds, produces an illusion of distance which even the knowledge of his presence can scarcely dispel. Thus, also, in the simple experiment with the cylinder; it is the easiest thing possible to deceive the person listening through the stethoscope, and to lead him to imagine the sources of the sound (*i. e.*, the points rubbed) to be approaching his ear, while they are actually receding from him, and *vice versâ*, such a result being produced by a simple variation in the force with which the different frictions are effected. Reasoning upon the facts and conclusions we have thus derived respecting the determination of the relative distances of sounding bodies by our sense of hearing, we may reasonably ask, whether intensity and loudness can be taken as sure guides to an accurate knowledge of the relative distances from the chest-wall of the sources of sounds heard in the cavity of the thorax. The experiments to which we have referred, incontestably show, that loudness cannot be depended upon as a measure of distance, and that a loud endocardial will appear more superficial, and nearer to our ear, than a weak pericardial murmur; and that very little reliance can be placed upon the apparent superficial or non-superficial character of a sound, as a means of unerringly distinguishing between an endo- and exocardial murmur. I fear that I have not explained my views as clearly as I might have done; but I have attempted to show that, as the relative distances of sounding bodies cannot be always determined by a comparison of their respective degrees of loudness, so the endo- or exocardial seat of a præcordial murmur cannot be always decided by the superficial character of the sound. If these views be correct, we return to the question, by what means are we to make an accurate diagnosis between the two classes of sounds?

The following considerations may assist us in making the distinction :—

(1.) As the auricular diastole occupies nearly $\frac{7}{8}$ths of the period of one beat of the heart, being divided in the proportion of $\frac{4}{8}$ths to the ventricular systole, $\frac{2}{8}$ths to the ventricular diastole, and $\frac{1}{8}$th to the period of ventricular repose. And as the auricular systole occupies $\frac{1}{8}$th of a beat of the heart, and occurs towards the end of a long silence, it follows that the walls of the auricles are either actively or passively in motion during the time the ventricles are at rest. A deposit of exudation upon the pericardium which covers the auricles, may, therefore, be very readily supposed to produce a friction sound during a portion of the ventricular repose; and hence the pericardial murmur, on close examination, is found commencing somewhat before the systolic ventricular contraction, and to be consequently not so synchronous with that movement as the murmurs which originate within the cavity of the heart.

(2.) For the same reason the pericardial murmur does not present the marked jet or gush-like sound which characterises the endocardial sound. It suggests the idea of a kind of rotatory or changing motion, rather than that of a fluid suddenly ejected from one cavity to another.

(3.) A pericardial, unlike some of the endocardial murmurs, is never propagated along the course of the great vessels.

(4.) A pericardial murmur is almost invariably continued through the periods of systole and diastole—an endocardial is not necessary double.

(5.) The cessation, reappearance, and ultimate disappearance of the pericardial murmur, contrasts strongly with the permanent character of the greater number of the abnormal sounds which result from organic disease of the valvular apparatus of the heart.

These considerations, added to the results of an examination of the præcordial region by the methods described in the previous lectures, will enable us to distinguish, in the majority of cases, a friction-sound due to pericarditis from a murmur resulting from endocarditis. At the same time, it must be admitted, that, in many cases, the diagnosis is exceedingly difficult, and sometimes impossible.

2. *Endocardial Murmurs.*

The nature of this important class of abnormal sounds may be learnt from considering the following conditions essential to their existence.

(*a.*) They originate, as their names imply, within the cavities of the heart, or the commencement of the great vessels which spring from that organ.

(*b.*) They may present every kind of variety of sound, such as blowing, rasping, filing, cooing, whistling, and musical. The French have named these varieties, *bruit de souffle, de râpe, de scie, sibilant musical,* &c.

(*c.*) They are exactly synchronous with the ventricular contraction or dilatation—one or both—they replace one or both of the normal systolic and diastolic sounds, and are heard in their greatest intensity at those parts of the præcordial region where the sounds in health are the easiest detected, viz., over the orifices of the aorta and pulmonary artery, and the apex of the heart.

(*d.*) They are caused either by,—

(*a.*) The presence of obstructions which impede the free flow of the blood through the heart and its great vessels, or by—

(*β.*) A supposed peculiar condition of the blood.

Let us briefly review the nature and extent of these causes.

(*a.*) And, in the first place, we find that the current of the circulation may be impeded by any cause capable of compressing the heart and its great vessels, or by the endocardium and valves becoming the seat of disease, and being rendered capable of fulfilling their proper functions. These causes may be termed compressive and obstructive.

The compressive, or those which reside external to the heart, are tumours in its immediate proximity; deformed and rickety chests which cramp and confine the space in which the organ is situated, the presence of fluid around the origin of the great vessels, &c. The circumstance of a murmur in the aorta and pulmonary artery being the direct consequence of a pericardial effusion, is deserving of more than a passing mention, as few writers have considered it in their description of the causes of the endocardial sounds. Zehetmayer fully explains the mode in which this variety of murmur is produced.

By an extension of pericarditis to that portion of the serous membrane which covers the commencement of the aorta, the coats of that vessel become penetrated by the exudation resulting from the inflammation. The elasticity of the parietes of the vessel being consequently impaired, the coats of the aorta are less disposed to yield to the force of the current injected in it from the ventricle. A slight impediment to the stream is therefore created, and a soft systolic bellows-murmur established. The presence of an accumulation of fluid around the base of the heart, by directly compressing the aorta, will necessarily favour the production of a similar sound which vanishes when reabsorption of the exudation has taken place. "The effusion contracts the impaired elastic tube of the aorta, and establishes an impediment to the current of the blood at the commencement of that vessel where its walls are thinned for the reception of the semilunar valves, and the circular and elastic fibres are deficient. A weak murmur will consequently arise there, as in arteries which are exposed to pressure. I have only observed this symptom in patients in whose pericardium a large quantity of fluid (1—1½ lbs.) was collected."* Another cause, external to the heart, is mentioned by Dr. Latham as sometimes productive of endocardial murmur—the too forcible application of the stethoscope over the præcordial region of young children whose chests are very thin and elastic. The space in which compression of this kind has caused the abnormal sound, is that part of the chest which corresponds to the arterial orifices. Dr. Latham states that he has never but in one instance produced a distinct murmur. He has, however, by pressing too heavily upon the præcordial region, often produced a kind of jarring sound simulating disease, and which has vanished with a gentler application of the instrument. I cannot say that I have had any experience of this cause of endocardial murmur.

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* Zehetmayer.—Die Herzkrankheiten, p. 124.

The obstructive causes, or those which reside within the heart and arteries, may be generally referred to an inflammation of the endocardium, and more especially of that portion of it which enters into the composition of the valves. As the object of these Lectures is to describe the method of detecting the results of disease, rather than to enter into a lengthened detail of the pathology of the heart, I shall simply mention the general course of valvular inflammation. This is found to be —

1. Redness, injection, and vascularisation of the fibrous structure of the valve.
2. Opacity, swelling and relaxation of the valve in consequence of,
3. Its infiltration by an inflammative exudation; and—
4. The deposition of granulations of coagulable lymph (vegetations) upon the edges and surface of the valves.

These may lead—

1. In some cases to the direct rupture of a valve or papillary tendon.
2. Sometimes to a purulent conversion of the inflammation, exudation, and to the subsequent softening and destruction of the valve.
3. Almost invariably to a permanent thickening and rigidity of the valve by the conversion of the exudation into fibrous tissue. Also to the contraction, rolling up, and shrinking of the valve, by which it is rendered incapable of performing its functions.

Looking at the results and consequences of valvular inflammation, so far as they relate to the orifices of the heart, we find that those outlets may be affected in a two-fold manner; either the stream of blood proceeding in its usual direction is compelled to traverse an abnormally contracted orifice, or else, in consequence of the imperfect action of the diseased valves, is partly made to retrace its course, or, in other words, to regurgitate through the deficiently closed orifices into the chamber from which it had been previously expelled. These morbid conditions of the orifices are termed—

1. Stenosis, or contraction of the orifice.
2. Insufficiency of the valves.

And to the resulting abnormal sounds, the terms direct and regurgitant murmur have been applied. As the orifices of the heart are four in number, and as each may be the seat of a direct and regurgitant murmur, eight endocardial murmurs are possible; but as the right side of the heart is very rarely the subject of valvular disease, and a direct mitral is a pathological curiosity, we have, in reality, only three varieties of endocardial murmur to study, viz—

Direct aortic,
Regurgitant aortic,
Regurgitant mitral,

the consideration of which I shall defer to my next Lecture.

LECTURE XVI.

AUSCULTATION.

At the last Lecture, I briefly sketched the causes, external and internal to the Heart, capable of producing an endocardial murmur, and I concluded with a reference to the changes induced by endocarditis in the orifices and delicate valvular apparatus of the organ. The subject is of such importance, that I shall not scruple to submit a Table contained in Zehetmayer's work, which brings together, in one view, the numerous internal structural lesions capable of causing an endocardial murmur. I have already stated the effects of disease upon the orifices of the heart to be twofold,—viz.:

(A.) Insufficiency—or an incomplete closure of the opening, and a consequent regurgitation or reflux of a portion of the blood-stream through the orifice.

(B.) Stenosis—or a contraction of the orifice, and a consequent tendency to impede the stream of blood which traverses the outlet.

According to the Table to which I have referred—

(A) Insufficiency may depend upon—

(*a.*) Disease of the tissue of the valves.

(*b.*) Disease of the papillary tendons,
(*c.*) „ „ papillary muscle,
(*d.*) „ „ parietes of the ventricles;

and analysing these divisions—

(*a.*) Disease of the tissue of the valves is observed in—

(1.) Shrinking and contraction of an entire valve, or the rolling up of its edges from the formation of a fibrous structure, or bony concretion within its substance.

(2.) Deposit of atheroma, proceeding to ulceration.

(3.) Rupture of the valve in several directions, chiefly from the loss of cohesion of its parts consequent upon an inflammation and infiltration of its tissue.

(4.) Perforation of the valves from an excess of atrophy.

(*b.*) Disease of the papillary tendons is observed in—

(1.) Thickening of the tendons from a deposit within their substance of a partially organised product of inflammation—they lose their glistening appearance and elasticity, become enlarged transversely and contracted longitudinally, and are consequently, rendered incapable of drawing the auriculo-ventricular valves completely across the corresponding orifices.

(2.) Union of several papillary tendons into one thick and non-elastic cord.

(3.) Attachment of the tendons to the under surface of the valves, by which the unfolding of the latter is impeded, and their free edges corrugated.

(4.) Rupture of one or more of the papillary tendons.

(*c*.) Disease of the papillary muscle is observed in—

(1.) Transudation and imbibition into its substance of an inflammatory exudation upon its surface, whereby the elasticity and contractile power of the muscle becomes injured and its capability of closing the auriculo-ventricular orifice impaired.

(2.) Deposit of a product of inflammation, and formation of fibrous substance in the papillary muscle, by which the fibres become atrophied and partially destroyed, and the entire muscle shortened.

(3.) Purulent degeneration of a deposited product of inflammation.

(4.) Fatty degeneration of the muscular fibres.

(5.) Rupture of a papillary muscle from loss of cohesion consequent upon inflammation, from the presence of abscesses, or from fatty degeneration.

(*d.*) Disease of the parietes of the heart is observed in—

(1.) The production of an excessively dilated orifice, which the valves, even when attenuated and enlarged, become incapable of completely closing.

(2.) A true aneurism of the heart in the proximity of the origin of a papillary muscle, will, of course, render that portion of the valve which is regulated by this muscle incapable of fulfilling its function.

(B) Stenosis. The causes of the contraction of an orifice may be—

(1.) Rigidity of the valves from the deposition of a product of inflammation within its substance.

(2.) Vegetations upon the surfaces and edges of the valves.

(3.) Union of the points of several valves producing a funnel-shaped or button-hole opening, through which the stream of the circulation has to force a passage. The mitral and aortic orifices may be reduced, in some cases, to mere chinks of scarcely 1—2 lines in diameter.

By these morbid processes a gradual atrophy is produced of the proper structure of the valve. A stenosis establishes in time an insufficiency of the orifice, while the converse is not always true.

The physical lesions which have thus passed before our view, are evidently sufficient to account for every variety of organic murmur capable of being detected in the interior of the heart, from the gentle souffle to the most acute, sharp, and whistling sound. These murmurs are manifestly the result of the vibrations into which the circulating blood is thrown in its passage through the various outlets of the heart, and depend upon—

(1.) Its friction against the roughness, vegetations, &c., of the valves.

(2.) The collision of its particles among each other in the rush of the stream through the contracted channels.

(3.) The rapid and forcible injection of a small quantity of blood into a chamber already partly filled with that fluid (regurgitation).

The character of the murmur—its softness or harshness—depends upon the nature of the abnormal surface its intensity varies with the force by which the current of blood is impelled through the obstructed outlet, and its duration with the time occupied by the movement of the blood-stream. I have already described the several varieties of murmurs which result from these conditions. Although the comparisons upon which their several appellations have been given are sufficiently correct, we must at the same time remember, that the varieties pass frequently into each other—a sound which is harsh and loud at one time, becoming soft and almost inaudible at another, when the action of the heart has slackened in speed and diminished in force.

In determining the orifice to which an endocardial murmur is to be referred, we have three principal circumstances to consider in aiding us to a correct diagnosis:

(1.) The position in the præcordial region at which the murmur exhibits its maximum intensity.

(2.) The period of the heart's action—its systole or diastole—in which it is heard.

(3.) The direction in which it may be traced over the surface of the chest.

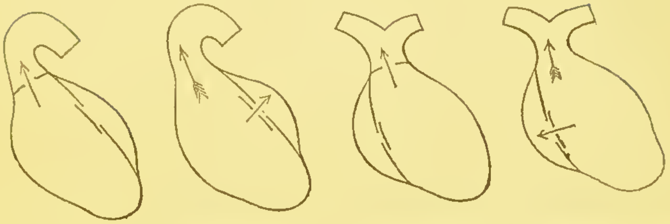
I have already mentioned that we have usually, in practice, only three different and distinct endocardial murmurs to consider, all of which originate in the left side of the heart, and may exist alone or in combination with each other. To make the subject, however, as complete as possible, and to facilitate the comprehension of the several actions going on within the heart at the time when any abnormal sound is produced, I refer you to the accompanying diagram, which embraces every variety of murmur capable of arising in either side of the organ and at any of its orifices.

The right and left sides of the heart are represented by different figures; the diseased orifice which produces the endocardial murmur is shown by a plain, and the healthy outlet by a feathered, arrow, the direction of which serves to exhibit the course of the blood-stream through the openings of the heart's chambers at the time of the occurrence of the murmur. In accordance with the diagram, we may classify the endocardial murmurs as follows:

1. SYSTOLIC	Left side of the Heart	Direct Aortic. Regurgitant Mitral.
	Right side of the Heart	Direct Pulmonic. Regurgitant Tricuspid.
2. DIASTOLIC	Left side of the Heart	Regurgitant Aortic. Direct Mitral.
	Right side of the Heart	Regurgitant Pulmonic. Direct Tricuspid.

As the space in which the four orifices of the heart are situated is bounded above by the lower edge of the cartilage of the third left rib; below by the lower edge of the cartilage of the fourth left rib; internally by the mesian line of the sternum; externally by an imaginary line at a distance of $1 — 1\frac{1}{2}$ from the central line of the body; and as this portion of the præcordial region corresponding to the orifices may be covered by the mouth of a stethoscope of $1\frac{1}{2} — 2$ inches in diameter, it is evident that the exact opening to which one or more of the eight possible murmurs is to

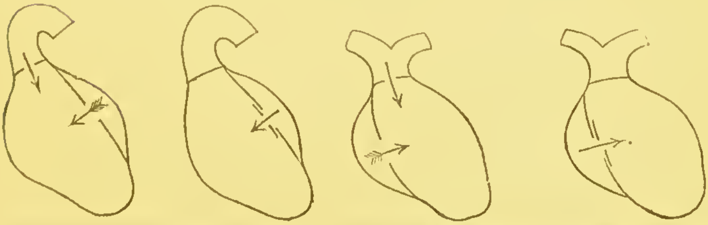
ABNORMAL MURMURS OF THE HEART.



Direct Aortic. Regurgitant Mitral. Direct Pulmonic. Regurgitant Tricuspid.

Left Side. Right Side.

SYSTOLIC.



Regurgitant Aortic. Direct Mitral. Regurgitant Pulmonic. Direct Tricuspid.

Left Side. Right Side.

DIASTOLIC.

be referred cannot be determined by simply confining our attention to this small division of the surface of the chest. If we remember, also, that the four orifices are not merely clustered together in a small space, but lie in such a manner that the valves of the pulmonary partly overlap those of the aorta, while the tricuspid opening is anterior to, and at no greater distance than one inch from, the mitral orifice, we shall still further see the difficulty of diagnosing the exact seat of a murmur from an examination confined to this valvular region. To localise the exact seat of the disease, we must observe the direction in which the murmur can be traced over the chest from its point of maximum intensity; whether it can be heard in the course of the aorta and the branches of that vessel, or only along the left edge of the sternum for a space of two or three inches; whether its intensity is excessive below the left nipple and extends to some distance downwards from that part, or over the lower and central portion of the sternum, almost as low as the epigastrium. To aid us still further in our diagnosis, we must examine the jugular veins, and discover whether they are simply swollen and gorged, or whether distinct pulsations can be traced along their course; and, lastly, we may take into our account the extreme rarity of disease of the pulmonic and tricuspid orifices, at least independent of a similar affection of the openings in the left side of the heart. "In truth, almost all our knowledge of endocardial murmurs proceeding from valvular disease is derived from our study of those which appertain to the left side of the heart." (Dr. Latham.)

Although the direct and regurgitant aortic and regurgitant mitral are the only murmurs ordinarily found in practice, I shall briefly describe the stethoscopic characters of the eight abnormal endocardial sounds represented in the diagram.

1. Aortic murmurs } Systolic or Direct.
(valvular) } Diastolic or Regurgitant.

(*a.*) The Direct Aortic is produced by the vibrations of the blood-stream in its passage from the left ventricle through a narrowed or roughened channel into the aorta. This sound may be usually traced in an upward direction towards the right edge of the sternum and between the cartilages of the second and third ribs of that side, passing frequently into the subclavian, carotid, and other arteries of the body. We observe from the diagram that the mitral orifice is closed, and the blood expelled in one direction only from the left ventricle, during the continuance of the murmur. As the vibrations are propagated with difficulty in a direction opposite to the blood-stream, the direct aortic murmur is very imperfectly heard over that portion of the chest which corresponds to the apex of the heart. It replaces the normal first sound, and is synchronous with the elevation of the pulse at the wrist and the stroke of the organ against the chest-wall. Its loudness is a measure, to a great extent, of the amount of friction to which the current of blood is exposed, and varies, therefore, with the quantity of the blood expelled at systole, and the force with which the current is driven over the friction-surface or through the narrowed orifice. A small amount of obstruction or roughness is occasionally attended by a much louder murmur than one of a more extensive nature, in consequence of the contractile energy of the heart being considerably greater in the former than in the latter case. Thus, in those extreme examples of aortic or mitral contraction in which the orifice has been reduced to a mere chink, the ventricle becomes incapable of propelling any considerable amount of blood from its cavity, and the resulting murmur is observed to be weak and imperfect. Hence, while the intensity of the murmur may be generally considered as a measure of the amount of the impediment, cases may occur in which the fact is exactly the reverse.

(*b.*) Regurgitant Aortic Murmur.

When the semilunar valves of the aorta are structurally incapable of closing the orifice of that vessel, upon the cessation of the systolic stream from the left ventricle, or when an abnormal dilatation of the commencement of the aorta renders the healthy valves insufficient for their function, a quantity of blood regurgitates during the diastole of the ventricle, and, coming into collision with a current of blood entering that chamber, produces the regurgitant aortic murmur. The friction of the stream against the sides of the orifice, and more especially against the edge of the imperfectly-closing valves, must aid very materially in the production of the sound. The position of maximum intensity will be the same as for the direct variety; and its point of origin, at the commencement of the system of blood-vessels, favours the transmission upwards along the aorta and its subdivisions. The murmur replaces the normal second sound of the aortic opening; but, by shifting the stethoscope slightly to one side, the sudden closure and flapping downwards of the valves of the pulmonary artery in diastole may be usually distinguished. An extreme amount of insufficiency, unattended by stenosis, may be recognised, in addition to the loud diastolic murmur, by a marked pulsation of the carotids, and a full, bounding, but rapidly retreating pulse—an observation originally made by Dr. Corrigan.

Disease of the aorta, whether due to a simple roughness of the lining membrane of the vessel or to a dilatation or partial contraction of its diameter, usually gives rise to a single or double murmur, which may be transmitted into the subclavian and carotid arteries. Such cases are not always to be easily distinguished from a structural alteration in the aortic orifice. Aneurismal dilatations of the vessel, when fully developed, occasionally produce a systolic impulse in the sternal region of the chest—a phenomenon of which I have already spoken in the Lecture upon Inspection. In the absence of this sign, our diagnosis will be materially assisted by a careful percussion of the aortic region.

2. Mitral Murmurs { Systolic or Regurgitant.
Diastolic or Direct.

(*a.*) Regurgitant Mitral Murmur.

The diagram shows us that the blood, during the occurrence of this sound, is expelled in two different directions at the same time from the interior of the left ventricle—normally through the aortic, and abnormally through the partially-opened mitral orifice. The friction of the abnormal current of blood against the edges of the auriculo-ventricular opening, and the sudden and forcible injection of a quantity of that fluid into the contents of the left auricle, give rise to a murmur which is synchronous with the ventricular contraction and the impulse of the apex of the heart against the ribs. The sound is rarely propagated into the aorta, but, being transmitted along the chordæ tendineæ of the mitral valve towards their points of attachment, is principally heard over the apex of the left ventricle, and may be in many cases traced to some distance below that point. It is also frequently detected in the axilla, and sometimes near the lower angle of the left scapula.

(*b*.) Direct Mitral Murmur.

In the healthy condition of the heart, the contents of the left auricle may be considered to gravitate rather than to be impelled with any considerable amount of force into the corresponding ventricle. The muscular parietes of the auricle are too thin and weak to produce any forcible expulsion of the blood contained in that chamber, which consequently glides smoothly onwards in its course with the evolution of no appreciable sound. An undoubted hypertrophy of the walls of the auricle, and a contracted condition of the mitral orifice, will even fail, in many cases, to produce a murmur, which is admitted by all observers to be the rarest of the morbid endocardial sounds.

A probable reason for its rarity may be found in the fact, that the current of blood to which it is due flows into the left ventricle through the mitral opening at the period of the heart's diastole, when the organ is at some distance from the chest-wall, and when any vibrations produced in the interior of the chamber cannot be expected to be communicated with much facility outwards. The systolic mitral murmur being produced at a time when the heart's apex is in actual contact with the parietes of the chest, is consequently more readily conducted to the ear of the auscultator.

The diastolic murmur may exist alone, or in combination with, the systolic murmur. "I have never met with more than one instance of the former. But a double murmur has been recognised at the apex in eighteen cases; out of these eighteen cases only four are known to have died—one during the period of observation, and three at subsequent periods. Eleven of them left the hospital (St. Bartholomew's) much relieved."*

My own observation would lead me to believe the diastolic mitral murmur to be even less frequent, as I cannot remember a single instance of simple diastolic mitral murmur. The sound is remarkable for the length of its duration, appearing in some cases to be almost continuous, and simply interrupted for a moment by the occurrence of the ventricular contraction. As already stated, it is usually associated with the regurgitant mitral murmur; and the structural lesion to which both sounds are due, usually occasions the production of a *fremissement cattaire*, or purring tremor.

3. Murmurs of the Pulmonary Artery $\left\{\begin{array}{l}\text{Direct.}\\ \text{Regurgitant.}\end{array}\right.$

An abnormal murmur capable of being referred to an organic alteration of the valves of the pulmonary artery, is a pathological curiosity, and not likely to come under our notice. The close proximity of the valves of the aorta and pulmonary artery, renders it difficult to determine the exact source of a murmur in their neighbourhood; but should the course of an abnormal sound be traced along the left edge of the sternum, and be suddenly lost at a distance of two inches from the base of the heart, and should it fail to appear in the subclavian or carotid arteries, we may fairly assume it to originate from the orifice of the pulmonary artery. These rules would be sufficient to establish the existence of an organic or functional disease of the vessel.

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* Dr. Ormerod, 'The Gulstonian Lectures,' 1851. 'London Med. Gazette,' p. 17.

I have already dwelt upon the excessive intensity of the normal second sound of the pulmonary artery in the case of an obstructed circulation through the left heart, and especially as resulting from organic alterations of the mitral valve. In consequence of the impediment, the current of the circulation becomes delayed, the left auricle gorged, the systems of the pulmonary artery and veins congested, the right side of the heart overfilled, dilated, and hypertrophied, the contractile energy of that chamber increased, the recoil of the pulmonary artery upon the contained blood unusually augmented, and the closure of the semilunar valves of the vessel effected with extraordinary force, and attended by the evolution of an excessively loud second sound.

4. Tricuspid Murmurs $\left\{ \begin{array}{l} \text{Direct,} \\ \text{Regurgitant,} \end{array} \right.$

are of equally rare occurrence as those of the pulmonary artery, and their diagnostic characters have not been fully determined.

A murmur over the central and lower part of the sternum, which is inaudible in the aorta and its branches, but conveyed downwards to the epigastrium, would fairly lead us to suspect its seat to be in the tricuspid opening. And a swollen condition of the jugular veins, and more especially a visible pulsation of these vessels at every ventricular systole, would confirm us in the belief of the existence of regurgitation through the right auriculo-ventricular orifice.

Functional Murmurs.—

The abnormal murmurs described have been considered to depend upon certain structural changes in the valvular apparatus of the heart, or in the lining membrane of the aorta,—changes which are usually beyond the power of medical treatment to remove, and which, consequently, impart a permanent character to the endocardial and vascular abnormal sounds. A less formidable class of murmurs is occasionally observed in the heart and great vessels (arterial and venous) which presents many points of resemblance to the organic variety, but differs essentially from the latter in its independence of structural lesion, and in being presumed by most writers to arise from a peculiar condition of the blood, and to disappear with the return of that fluid to its proper conditions of quantity and quality. These murmurs are termed functional, in contra-distinction to the organic and anæmic or chlorotic, from the supposed peculiar condition of the blood to which they are usually ascribed. We shall observe hereafter the amount of credit to be attached to the latter view of the origin of these sounds. These murmurs may be divided into—

1. Venous { Jugular.
  Sternal.

2. Arterial.

1. *Venous Murmurs.*—

I commence with this variety, for the purpose of commenting upon a collection of observations which I have made upon a large number of individuals of both sexes, and of all ages, and from which several important results may be deduced.

If the stethoscope be placed over the jugular vein of an individual in whom the skin of the neck is tolerably thin, free from stiff corrugations, and unencumbered by subjacent fat,—conditions chiefly to be found in children, youths of both sexes, and in anæmic and chlorotic persons,—a continuous or occasionally interrupted hum, cooing, or even whistling sound may be heard, which varies in intensity from time to time, and presents every degree of loudness, from a gentle souffle to a loud, roaring murmur. To this sound the French and German writers have applied the terms of *bruit de diable* and *Nonnengeraüsch* respectively.

Again, if the instrument is applied to the upper bone of the sternum, and towards the right sterno-clavicular articulation,—a sound precisely similar in character, but of much weaker intensity, may be frequently detected, to which I shall take the liberty of giving the name of the Sternal Venous Murmur. The position at which this sound is heard corresponds to the junction of the right and left venæ innominatæ, and its origin may be partly referred to the molecular collision of the two streams of blood at the moment of their union to form the column of the superior vena cava. Its intensity is usually weak, and easily unobserved, in consequence of the predominance of the normal respiratory murmur; but, in some cases, its loudness is remarkable, and even whistling in character. Its presence will, of course, be more readily detected by causing the individual under examination to cease breathing for a few moments. In some instances I have been able to follow the sound in a downward direction along the course of the vena cava; but, as already stated, its seat is the upper bone of the sternum, and all trace of it is usually lost when the stethoscope is removed towards the orifices of the aorta and pulmonary artery.

Considerable contrariety of opinion has been expressed respecting the nature of the continuous murmur heard in the neck. At the period of its discovery, and even at the present time according to some writers, (Professor Kiwisch for example,) the seat of the sound was supposed to be the carotid artery; but I believe that no doubt can be entertained of the correctness of the view which originated with Dr. Ogier Ward of the true seat of the murmur being in the jugular vein, and for the following reasons:

1. The continuous murmur is frequently found to be coexistent with a distinct carotid impulse, sounds which are evidently incapable of existing at the same moment in the same vessel.
2. The murmur is interrupted by pressing with the finger upon that part of the jugular vein which is situated above the stethoscope, an effect not observed in the sound of the accompanying carotid impulse.

3. The two murmurs may be occasionally heard separately by employing a very small-ended stethoscope, and shifting it slightly, right or left.

4. The murmur is increased in loudness by any cause which accelerates the flow of the blood through the jugular vein, and *vice versâ*. Thus :—

   (*a.*) The act of inspiration, by its favouring the current of the blood to the right side of the heart, augments the loudness of the continuous murmur. The reverse occurs during the act of expiration.

   (*b.*) The murmur becomes weak and usually inaudible when the individual under examination is made to assume the recumbent posture. It is curious to observe the gradual change in the intensity of the sound with the gradual inclination of the body. The diminution and cessation of sound can only be referred to the loss of velocity, caused by the change from the perpendicular to the horizontal position. The change of inclination has no perceptible effect upon the carotid impulse.

   (*c.*) The right is smaller in calibre, but more perpendicular in its course, than the left jugular vein. Now, as we have no reason to believe that there is any difference between the quantities of blood returned to the heart through the two vessels, it follows that the stream in the right must be quicker than the current in the left vein. Hence, with the greater friction, we should naturally expect a louder, or at least a more frequent, occurrence of the sound in the right than in the left side of the neck. Our observations prove this view to be correct.

(*d.*) The murmur is never heard when the jugular veins are swollen and turgid, or when the stream through them is delayed from any cardiac obstruction.

(*e.*) The details of the following case, related in Schmidt's 'Jahrbücher' for 1850, by Dr. Richter, appear to confirm this view conclusively. In consequence of an obliteration of the right jugular vein, the right external mammary had not only become dilated to the size of a goose-quill, but had established a collateral communication with the internal mammary vein of the left side. At the points where the dilated branches passed through the fourth, fifth, and sixth intercostal muscles, a clear and almost musical continuous murmur was distinctly audible. In this case no arteries were present to which the sound could be referred.

Having thus shown that the cause of the murmur must be sought for in the venous system, we have, in the next place, to determine the nature of the cause, and the meaning of the sound. The majority of writers upon the subject suppose it to depend upon a peculiar watery condition of the blood, attended by a marked diminution in the number of the corpuscles, and in the amount of colouring matter. And, according to the views of M. Andral, the murmur is invariably audible whenever the number of the blood-corpuscles falls from the normal 127 per 1000 parts of blood to 80 per 1000, and lower. Hence the sound has been termed an anæmic or chlorotic murmur *par excellence,* and its appearance has been considered to be a direct indication of the necessity of administering some preparation of iron to the individual in whom it is detected. The observations which I have made upon more than 1000 persons of all ages, entirely negative this exclusive view of the cause of the murmur, inasmuch as they prove that the sound is not only present in the pale, anæmic, and chlorotic girl, but also in individuals of both sexes (particularly the young), who exhibit every appearance of strong and ruddy health.

Having seen a statement made by Dr. Liman, of Berlin, of the remarkable frequency of the venous murmur in children, I determined to examine a large number of young persons; and, by the courtesy of the medical and official authorities, I was enabled to pursue my inquiries at the Infant Orphan Asylum, Wanstead; the Merchant Seamen's Orphan Asylum, Mile-end Road; the Union and Park House, Clapton; and the London Orphan Asylum; and, to test still further the correctness of my conclusions, I selected for examination a number of fine, rosy children at Limpsfield and Farnham, in Surrey. My observations were particularly directed to the absolute and relative frequency of the right and left jugular murmurs, and to the number of instances in which the sternal venous sound could be detected: and I did not forget to examine the præcordial region for the appearances of valvular functional murmurs.

The table on the opposite page gives the analysis of the examination of 802 healthy children, from 14 months to 15 years old:*

Having proved the extreme frequency of venous murmur in children, I determined to pursue the subject still further. For this purpose 100 healthy young men, varying between 17 and 23 years of age, belonging to the Provisional Battalion, were kindly placed at my disposal by the Commandant at Chatham. Although the majority of these soldiers presented a ruddy complexion, the following were the results at which I arrived:—

Venous murmur loud on both sides				44
Right venous murmur	loud	left venous murmur	weak	16
" "	weak	" "	loud	11
" "	distinct	" "	absent	3
" "	absent	" "	distinct	1
Traces on both sides				10
Absent on both sides				15
				100
Sternal venous murmur present in				11

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* For the examination of children, a stethoscope must be employed which has a small end, not exceeding the size of a sixpence. The want of this precaution, has probably caused the frequency of the venous murmur in children to have been overlooked by former observers. The investigation of the murmur in adults must be also conducted with some address, as the sound will be observed only in certain positions of the neck, and with certain degrees of pressure, to be found by repeated trials.

		Wanstead Orphan Asylum.	London Orphan Asylum.	Merchant S. Orphan Asylum.	Hackney Union.	ParkHouse Clapton.	Limpsfield, Surrey.	Farnham, Surrey.	Total.
Venous Murmur loud on both sides	Male	77	68	40	25	31	21	14	276
	Female	84	52	27	38	28	20	0	249
Right, loud; left, weak	Male	31	15	6	2	7	3	4	68
	Female	33	18	8	3	3	6	0	71
Right, weak; left, loud	Male	14	10	2	3	3	3	1	36
	Female	9	2	3	3	1	1	0	29
Right, distinct; left, absent	Male	2	0	1	1	1	1	0	6
	Female	4	5	3	0	0	2	0	14
Right, absent; left, distinct	Male	0	0	0	0	0	0	0	0
	Female	1	0	0	0	0	0	0	1
Traces on both sides	Male	6	4	0	0	1	0	0	11
	Female	0	6	0	3	4	1	0	14
Absent on both sides	Male	13	5	0	0	1	0	1	20
	Female	8	7	1	0	0	1	0	17
Total	Male	143	102	49	31	44	28	20	417
	Female	139	90	42	47	36	31	0	385
		282	192	91	78	80	59	20	802
STERNAL VENOUS MURMUR.									
	Male	29	18	5	6	10	7	3	78
	Female	36	10	6	10	7	5	0	74
Total		65	28	11	16	17	12	3	152

An examination of 53 healthy females, at the Female Asylum, Dalston, and at St. Luke's Union, gave the following result. These persons varied from 16 to 28 years of age.

Venous murmur loud on both sides				17
Right venous murmur	loud	left venous murmur	weak	13
,, ,,	weak	,, ,,	loud	3
,, ,,	distinct	,, ,,	absent	3
,, ,,	absent	,, ,,	distinct	1
Traces on both sides				9
Absent on both sides				7
				53
Sternal venous murmur present in				8

Subsequent inquiries showed that the venous murmurs became less frequent after the prime of life, although the jugular may be sometimes heard in extreme old age. Between the ages of 30 and 60, I have, in many instances, found the continuous murmur in the neck, but the sternal sound in only one or two cases. I have never been able to detect the latter in persons beyond 60 years of age.

The following table gives the result of an examination of 67 old people of both sexes:

Females	5 from	50—60 years of age	1 right ven. mur.	4 absence of sound.
,,	17 ,,	60—70 ,,	1 ,, ,,	16 ,,
,,	24 ,,	70—80 ,,	2 ,, ,,	21 ,,
,,	,,	,,	1 right & left v. m.	– ,,
,,	2 ,,	80—90 ,,	–	2 ,,
	48		5	43
Males	2 ,,	50—60 ,,	. . . .	2 absence of sound
,,	6 ,,	60—70 ,,	2 right ven. mur.	4 ,,
,,	11 ,,	70—80 ,,	1 ,, ,,	10 ,,
	19		3	16

In none of these latter cases, male or female, was the sternal venous murmur present.

The facts which I have collected will, I think, allow us to establish the following conclusions:

1. That the venous murmur does not necessarily depend upon any abnormal condition of the blood, nor upon any deviation from the health of the individual in whom it may be found, for we have observed it to be almost universal in children, to be present in a large proportion of persons under the age of 25 years, and to exist occasionally in the aged—all in the most perfect health. It is not, therefore, an anæmic or chlorotic murmur, although uniformly present in those conditions of the system which are marked by an impoverished condition of the blood, inasmuch as it has been observed in a multitude of instances to coexist with the ruddiest complexion and the most perfect health.

2. That the venous murmur is not entirely the result of pressure, although some portion of the sound may be fairly attributed to that cause. The existence of a sternal venous murmur at a spot upon which no pressure can be exerted by the stethoscope, is a sufficient proof that sound can originate in the venæ innominatæ, independent of any compressing cause; and if in these veins, why not in the jugulars also? If, then, these murmurs can neither be attributed to the transit of thin and impoverished blood through the veins, nor to the effects of external compression upon the parietes of these vessels, in what mode are we to explain their origin? I believe very easily. There can be no doubt that the rapidity of the blood in the large veins is usually sufficient to establish a friction capable of causing a

sound, which is more or less audible according to the readiness with which the parietes of the veins take up the vibrations, and the facility with which the latter are conducted to the outer surface of the body. The three elements in the production of the murmur in healthy individuals are, therefore,—

(1.) A certain velocity of circulation;
(2.) An elastic condition of the parietes of the vein;
(3.) A good conducting medium between the vein and the surface;

the imperfection of any of which will produce a corresponding diminution in the resulting murmur. The sound is of such frequent occurrence in the healthy child, in consequence of the rapidity of its circulation, the thinness of the parietes of the veins, and the elastic nature of the skin and its subjacent structures. The same reasons apply with equal force to the chlorotic girl, whose "sharp knocking heart" indicates an amount of ventricular contraction sufficient to produce an abnormal velocity in the general current of the circulation. The thin and impoverished condition of the blood, which is an undoubted condition of chlorosis, will also tend to the maintenance of the velocity and to the production of an unusual friction in the veins.* The increase of age brings with it a diminution in the rapidity of the pulse, a thickened or corrugated condition of the parts around the vein, and a probable alteration in the parietes of that vessel, by which its elasticity becomes impaired. To these causes may, perhaps, be added a general diminution in the circulating mass. Hence the unfrequency of the sound after the middle period of life.

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* The fluid portion may increase in Chlorosis, Scurvy, and Bright's disease, from the normal 775 to the abnormal 870, 849, and 880 in 1000 parts by weight of blood, (Simon's Chemistry). Although blood of such diminished density might be supposed *cæteris paribus* to be more readily thrown into vibration than healthy blood, still the venous murmur

Such are the conclusions to which I think we may safely arrive from an attentive consideration of the facts which have passed under our observation. I might occupy your time with the numerous theories which have been advanced in explanation of the origin of these murmurs. I might dwell upon the view of Vernois, who supposes that the veins become relaxed in anæmia, and consequently present folds of their internal lining, which obstruct the stream of blood and produce the sound. I might enter into the arguments, *pro* and *con,* with respect to the latest theory of the talented observer, Hamernjk, who believes that in spanæmia the thin rill of blood imperfectly fills the jugular vein, and runs downwards towards the heart whirling and gurgling and producing murmurs. I might quote Barth and Roger, who ask "why venous murmurs are not produced in the veins during the state of health," and who satisfy themselves with the experiments of M. Aran, and the conclusion at which he has arrived, that "the intensity of the murmur is always in the inverse ratio of the density of the blood." But such a review would be useless, as the results at which our observations have arrived com-

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in such cases is frequently not so loud as in healthy and ruddy individuals, in whom the current of the circulation is maintained in full vigour by a firmly contracting heart. At the same time Hydræmia must be considered to be a predisposing cause of murmurs, venous as well as arterial.

pletely removes the very basis upon which these explanations are founded. The murmur is not confined to cases of anæmia, spanæmia, or chlorosis: it is a very frequent accompaniment of the most robust health, and possesses, therefore, no special value as an indication of disease.

Arterial Functional Murmurs.

A few remarks will comprehend all I have to say respecting this class of murmurs. That such sounds do exist cannot be doubted; but my experience leads me to the opinion, that a murmur, which originates in the proximity of the semilunar valves or commencement of the aorta—a real bellows-sound, independent of organic lesion, is a much less frequent occurrence than is generally supposed. I know not why the arterial should not be as often observed as the venous functional murmurs, unless the walls of the arteries, from their greater thickness, are less prone to take up vibrations than those of the veins;* but of the vast disproportion in the comparative frequency of the organic and functional arterial abnormal sounds, I am fully convinced. That loss of blood will produce a bellows murmur, has been proved by the experiments of Dr. Marshall Hall upon dogs. The same fact may be observed in the human subject in cases of menorrhagia, or when a considerable quantity of blood has been lost from some serious bodily accident. Anæmic and chlorotic individuals occasionally exhibit a præcordial *bruit de souffle;* but from the notes which I have made of this class of patients, I am led to believe, that the number in which the murmur is present, bears a very small proportion to those in which it is absent. The following may be taken as a brief summary of the peculiarities of the functional murmur:—

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* The relative thickness of the parietes of the jugular vein and carotid artery, I have found to be nearly as seven to twenty. The most accurate method of obtaining this result is to place together an equal number of thicknesses of each vessel, and to compare the measurements of the two collections. We shall find the coats of the jugular vein to be about one third of the thickness of those of the carotid artery, a difference sufficient, in some measure, to acount for the greater frequency of the murmur in the vein than in the artery.

1. It is exclusively situated at the base of the heart, and never propagated along the course of the aorta.
2. It coincides uniformly with the systole of the ventricles, and is never heard during the dilatation of those chambers.
3. Its tone is usually (though not universally) soft and blowing.
4. It is remarkable for its intermittence, and its ultimate disappearance.

The peculiar restriction of the sound to the base of the heart, and its absence in the ascending aorta, have, for some time past, induced me to suspect the real seat of the murmur to be in the pulmonary artery; an opinion expressed also by Dr. Hughes in a paper published on Anæmic Murmurs, in the 'Guy's Hospital Reports' for 1850. Further observations are required to establish this view. Can a plethoric condition of the body produce a functional murmur? I know of no proofs to substantiate the idea, although Barth and

Roger assert such a course to be possible. Is an excessive action of the heart capable of eliciting the sound? Dr. Latham relates the case of a young woman in perfect health, who was seized with a violent hysterical emotion upon the death of her infant, and in whom the loudest possible præcordial bellows-murmur was audible, so long as the contractions of the heart were forcible and violent.

A French physician Dr. Jacquemier published, in 1837, the result of his examination of a number of females before, during, and after pregnancy, and his statements have been copied into recent works upon auscultation. He found the bellows-murmur present,

In $\frac{1}{4}$th of those examined during pregnancy,
„ $\frac{1}{6}$th „ „ immediately after delivery,
„ $\frac{1}{36}$th of non-pregnant females.

I have frequently attempted to verify this statement, but without success, and I can find no work in which this statement has been confirmed. I have merely alluded to the observation, as I find it adopted in the valuable treatises of Barth and Roger, Zchetmayer, and Skoda, although no mention is made of the personal experience of these authors upon the subject.

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